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In some tables, the designations "developed" and "developing" economies are intended for statistical convenience and do not necessarily express a judgement about the stage reached by a particular country or area in the development process.

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PREFACE

The purpose of the *Population Bulletin of the United Nations,* as stipulated by the Population Commission, is to publish population studies carried out by the United Nations, its specialized agencies and other organizations with a view to promoting scientific understanding of population questions. The studies are expected to provide a global perspective of demographic issues and to weigh the direct and indirect implications of demographic policy. The *Bulletin* is intended to be useful to Governments, international organizations, research and training institutions and other bodies that deal with questions relating to population and development.

The *Bulletin* is prepared by the Population Division of the Department of International Economic and Social Affairs of the United Nations Secretariat and published semi-annually in three languages—English, French and Spanish. Copies are distributed widely to users in all member countries of the United Nations.

Although the primary source of the material appearing in the *Bulletin* is the research carried out by the United Nations Secretariat, officials of governmental and non-governmental organizations and individual scholars are occasionally invited to contribute articles.

CONTENTS

Explanatory notes

Symbols of United Nations documents are composed of capital letters combined with figures. Mention of such a symbol indicates a reference to a United Nations document.

Reference to "dollars" ($) indicates United States dollars, unless otherwise stated.

The term "billion" signifies a thousand million.

Annual rates of growth or change refer to annual compound rates, unless otherwise stated.

A hyphen between years (e.g., 1984-1985) indicates the full period involved, including the beginning and end years; a slash (e.g., 1984/85) indicates a financial year, school year or crop year.

A point (.) is used to indicate decimals.

The following symbols have been used in the tables:

Two dots (..) indicate that data are not available or are not separately reported.

A dash (—) indicates that the amount is nil or negligible.

A hyphen (-) indicates that the item is not applicable.

A minus sign (-) before a number indicates a deficit or decrease, except as indicated.

Details and percentages in tables do not necessarily add to totals because of rounding.

The following abbreviations have been used:

CELADE	Centro Latinoamericano de Demografía
CICRED	Comité international de coopération dans les recherches nationales en démographie (Committee for International Co-operation in National Research in Demography)
CMEA	Council for Mutual Economic Assistance
ESCAP	Economic and Social Commission for Asia and the Pacific
ECE	Economic Commission for Europe
ECLAC	Economic Commission for Latin America and the Caribbean
ESCWA	Economic and Social Commission for Western Asia
FAO	Food and Agriculture Organization of the United Nations
ISI	International Statistical Institute
IUSSP	International Union for the Scientific Study of Population
OECD	Organisation for Economic Co-operation and Development
UNDP	United Nations Development Programme
UNFPA	United Nations Fund for Population Activities
WFS	World Fertility Survey

A SIMPLIFIED PRESENTATION OF COHEN'S MODEL ON ESTIMATING THE EFFECTS OF SUCCESSFUL MALARIA CONTROL PROGRAMMES ON MORTALITY

United Nations Secretariat *

SUMMARY

This paper presents a general overview of the method developed by Joel Cohen to estimate the effects of successful malaria control programmes on mortality. His approach is based on the possibility of separating a population into certain groups according to the disease-specific mortality risk and on how a given intervention will differentially affect each of these groups. The Cohen model can be used as a tool to project the minimum and maximum impact on mortality under different assumptions of the success of a programme, given the basic nature of the programme and the current situation of the disease.

INTRODUCTION

In this article we consider the estimation of the expected number and age-sex distribution of deaths averted by a health intervention for one specific disease: malaria. The article describes in summary fashion a model developed by Joel Cohen, in co-operation with the Population Division, Department of International Economic and Social Affairs, United Nations Secretariat, for measuring the expected effect of a successful malaria control programme on human mortality levels and structure.** Although the model is specific to malaria control programmes, it appears generalizable and we hope it will inspire the construction of similar models for other diseases.

Diseases transmitted by insects and other vectors are widespread in tropical areas of developing countries and have serious adverse consequences on the population. Malaria remains the most prevalent of such diseases. In 1986, the WHO Expert Committee on Malaria estimated that over 2 billion persons live in "areas where a reduced level of infection is maintained by the continued application of antimalarial measures". In addition, 365 million persons reside in malarial areas where "no organized control measures exist". These two figures combined represent nearly one half of the world's population. Most malarious countries of Asia and the Americas are included in the former category, and those of Africa south of the Sahara in the latter. The World Health Organization (WHO) also estimated that the world incidence of malaria is nearly 100 million cases annually, and that this situation had not changed during the past 15 years (World Health Organization, 1986a, p. 9). Although malaria prevalence has declined in some developing countries, notably China and India, the malaria situation has deteriorated elsewhere, particularly in rural areas of Asia and the Americas, due to both the increasing resistance of mosquito vectors to insecticides and of some malaria parasites to available drugs. For example, chloroquine-resistant *P. falciparum* malaria has now been found in more than 40 countries (World Health Organization, 1986b).

On the other hand, recent advances towards the development of a malaria vaccine have raised hopes in many quarters of greatly reducing the prevalence of malaria. Therefore, it is of considerable importance to investigate the effects of successful efforts to reduce malaria on the mortality levels and structure to help Governments monitor their intervention programmes, to increase understanding of the determinants and consequences of mortality conditions, and to project mortality as an input to overall population projections.

The Cohen approach is based on the possibility of separating a population into certain groups according to the disease-specific mortality risk, and how a given intervention will differentially affect each of these groups. The model can be used as a tool to project the minimum and maximum impact on mortality under different assumptions of the success of a programme, given the basic nature of the programme and the current situation of the disease. It then provides an input for the monitoring of malaria control programmes (mcps) by allowing field workers to analyse, decompose and evaluate the achievements of a programme in order to draw implications for further implementation and modification.

The model is notable for its mathematical simplicity, which masks the complexity of the epidemiology of malaria which is explored in detail in the Cohen article. It is essential for the field epidemiologist who is applying the model to study carefully the mathematical relationships presented in the article by Cohen to understand

* Population Division, Department of International Economic and Social Affairs.

** See "Estimating the effects of successful malaria control programmes on mortality" in the present publication.

their underlying assumptions. Some readers may be interested in a general overview of the model and its potentials. For them, we have prepared this short accompanying document which attempts to explain the spirit and rationale of the model in a less technical way. We hope this short document will, therefore, bridge a potential gap between health policy makers and field technicians.

Before continuing, four additional points must be made. First, as in any effort to measure the expected effects of a mortality control programme, this model considers not only lives saved directly from controlling the disease of interest but also those saved indirectly due to the synergistic relationships and the competing risks among diseases. Secondly, the model only attempts to measure the expected effects of the intervention on mortality; morbidity is not considered. Thirdly, the model does not intend to estimate the impact of exogenous changes (factors other than the malaria control programmes) on mortality. Lastly, throughout these two papers, we speak of the "success of a programme", and study the expected mortality effects under assumptions of the "degree of successfulness". "Success" in these papers indicates the extent to which a programme has attained its goals with respect to programme delivery; for example, the extent to which the area of interest has been covered by spraying and the mosquito eradicated, or to which fever cases have been treated by chemotherapy.

The model

In this section we present the fundamental elements of the Cohen model. As will be seen, there are four key issues addressed in the model, namely: (*a*) how the total population is distributed according to malaria risk; (*b*) how the death rate of the total population is decomposed into different groups according to the malaria status; (*c*) what kind of information is required and what kind of assumptions must be made on the success of mcps; and (*d*) how much is mortality reduced in each subpopulation given the assumed success of the different types of programmes.

Distribution of the population according to malaria status

Fundamental to the model is the division of the malaria-affected human population into four subpopulations (or groups): fever cases, currently infected (blood cases), previously infected (immune cases) and never infected. The population of these four groups is mutually exclusive and fully inclusive in terms of malaria infection.

These four subpopulations are defined both clinically and operationally. The operational definition for each group must take into consideration the robustness of the various tests that are undertaken in malaria surveys, that is, their risk of false positives and false negatives. In light of the above, the suggested useful operational definitions are as follows:

(*a*) "Fever case", denoted by f, is defined as a person, living in or coming from a malarious area, with a temperature of at least 38°C (or 100.4°F) associated with malaria, and with no other apparent cause of illness. Chills, sweats or other signs and symptoms consistent with malaria may also occur but are not required for a person to be identified as malarial. According to Cohen, the criteria used to define fever cases may be workable in the field where microscopy is not possible;

(*b*) "Currently infected (blood case)", denoted by b, is defined as someone with malarial parasites determined by microscopic examination of the blood (blood test) but who currently exhibits a temperature of less than 38°C or 100.4°F;

(*c*) "Previously infected (immune case)", denoted by i, is a person who gives evidence of prior infection with malaria, either by enlargement of the spleen, or by the presence of anti-malarial antibodies in the blood serum, and who is not currently infected or a fever case;

(*d*) The never-infected subpopulation is the residual, denoted by 0. These are the people who exhibit no evidence of malarial infection by the blood test and do not have fever or an enlarged spleen.

Expected change in the death rate of the total population

The model proposed by Cohen estimates the expected change in the death rate of the total population due to the successful mcp by projecting the proportion of the population within each subpopulation with respect to the infectious status and death rates of those subpopulations after the intervention.

The proportions of the four subpopulations before the mcp are p_f, p_b, p_i and p_0; those after the mcp are p_f', p_b', p_i' and p_0'. Death rates of the subpopulation before the intervention are d_f, d_b, d_i and d_0; they are d_f', d_b', d_i' and d_0' after the intervention. The total death rate for the population before intervention (d) is decomposed as

$$d = (p_f d_f) + (p_b d_b) + (p_i d_i) + (p_0 d_0), \qquad (1)$$

that is, the total death rate of a population is the sum of the death rates of different groups weighted by the proportion of the population in those groups. Similarly, the total death rate for the population after a successful mcp (d′) can be decomposed as

$$d' = (p_f' d_f') + (p_b' d_b') + (p_i' d_i') + (p_0' d_0') \qquad (2)$$

The model rests on the presumption that, where the prevalence of malaria is considerable, there are substantial differences in the death rates between the four subpopulations, and that the mcp, if implemented successfully, will reduce those differences significantly.

Information required and assumptions about success of the programmes

In order to estimate the expected change in the total death rate of a population ($d - d'$) due to a given type of programme, information is necessary on death rates of each subpopulation, and the proportion of each subpopulation in the total population (weight). Besides, assumptions about the success of the mcp and about exogenous mortality changes must be made.

First, information on the distribution of the population among the groups (p values) and their death rates (d values) can be and usually are collected in malarial surveys, although the required tabulations are rarely made. The expected reduction in the death rate of the total population due to the mcp depends heavily on these values. For example, if the death rates of the first three groups are about the same as the death rate of the never-infected group, the programme is unlikely to produce a significant mortality decline. Or if the proportion of fever cases and the currently infected cases is very small, a reduction of their death rates will not have significant impact on the death rate of the total population.

Secondly, assumptions need to be made about the extent to which the programme will succeed. The extent of success is represented by a set of success coefficients whose values range from zero (complete failure) to one (complete success). Alternative sets of values of the success coefficients may be used for different scenarios of the programme performance.

Thirdly, presumed changes in mortality due to factors other than the intervention need to be specified. The change of the death rate from before to after an intervention will be due to both the malaria control programme and exogenous changes in mortality which are unrelated to the intervention programme. The model estimates expected changes in the proportion of each subpopulation and in the death rate of each subpopulation due to the mcp. The effect of the exogenous changes are specified outside the model. Such an exogenous change may be determined by observation of the change in a control group, by extrapolation of recent changes, or by other assumptions, such as a change consistent with an improvement in life expectancy at birth of 0.5 years per year.

Lastly, the type of mcp must be determined since the mortality effect will correspond to the programme type. Three types of malaria control programmes are considered by Cohen: chemotherapeutic programmes, multifaceted programmes and eradication programmes. Under chemotherapeutic programmes, patients (fever cases) are treated with antimalarial drugs. Multifaceted programmes include not only chemotherapy, but also limited chemoprophylaxis and low-cost vector control. Under multifaceted programmes, the risk of death of both fever cases and currently infected cases may be reduced because of chemotherapy and chemoprophylaxis. The proportion of the subpopulation that has never been infected may increase, and the proportions of fever and currently infected cases may decrease, due to vector control. An eradication programme builds on a multifaceted programme by adding expanded vector control operations, including continued surveillance to ensure maintenance of the eradication. With a successful eradication programme, the entire population in the treatment area will eventually move into the never-infected group as the pre-mcp cohorts die out.

Changes according to type of intervention programme

Chemotherapeutic programmes. The change of death rate due to chemotherapeutic programmes can be explained as follows. These programmes only affect the subpopulation of fever cases. The proportion of the fever cases (p_f) is assumed to stay the same because it is likely that persons who were treated with drugs would be reinfected (drugs prevent death from malaria). Under this programme, previously infected individuals are not affected; they show no clinical signs of acute malaria so they are not treated.

Cohen defines the success coefficient (s_f) as a fraction of the fever cases who are successfully treated by a chemotherapeutic programme. It falls between 0 and 1. Therefore, a chemotherapeutic programme that successfully treats fraction (s_f) of the fever cases would change the excess mortality from ($d_f - d_0$) to

$$d_f' - d_0' = (1 - s_f)(d_f - d_0). \qquad (3)$$

If all fever cases are treated successfully ($s_f = 1$), the post-mcp death rate of fever cases is simply d_0', the death rate of the never infected. If none are treated successfully ($s_f = 0$), the excess mortality after the intervention equals the excess mortality before the mcp.

In reality, persons who are fever cases and saved from dying from malaria may die of other diseases. For the sake of simplicity, the effect of the competing risk is not incorporated here.[1] From equation (3) it can be easily seen that the post-mcp death rate for the fever cases equals the post-mcp death rate for the subpopulation of never infected, plus the excess mortality of the fever cases from malaria before the intervention, multiplied by the complement of the success coefficient.

Cohen points out that the distribution of the population by malaria infection status will remain unchanged under a chemotherapeutic programme, because chemotherapy prevents reinfection of malaria only for a negligible period of time. Since only fever cases are affected, death rates of other groups remain unchanged. So the mortality decline in the population due to the chemotherapeutic malaria control programme ($d - d'$) is simply the difference between $p_f d_f$ and $p_f d_f'$ in the absence of an exogenous change. It can then be easily derived from equations (1), (2) and (3).

Multifaceted programmes. In these programmes the death rates of both the fever cases and currently infected cases (blood cases) are reduced. Post-programme excess mortality for fever cases ($d_f' - d_0'$) will be as shown above [equation (3)] and the excess mortality for the currently infected ($d_b' - d_0'$) similarly will be

$$d_b' - d_0' = (1 - s_b)(d_b - d_0). \qquad (4)$$

Different success coefficients (s_f and s_b) can be assumed for the fever cases and currently infected cases (blood cases), respectively. However, often there will be no evidence of s_f and s_b being different so they can be assumed to be identical.

In contrast with a programme which is solely chemotherapeutic, in a multifaceted programme the proportions of the subpopulation of fever cases and currently infected cases will decline. After the malaria control programme, the proportion of the subpopulation of fever cases (p_f') and currently infected cases (p_b') is a

function of effective success factors similar to the ones applicable to the death rates. The post-mcp proportions of the groups of fever cases and currently infected cases are estimated as

$$p_f' = (1 - s_F)p_f \quad (5)$$

and

$$p_b' = (1 - s_B)p_b, \quad (6)$$

where s_F is "the fractional reduction in the size of the subpopulation of fever cases", and s_B is "the fractional reduction in the size of the subpopulation of currently infected cases" (see p. 16).

It is necessary to mention here the differential effect of the programme on the population of younger individuals (0-4 age group) and the older ones (ages 5 and older). As noted by Cohen: "For the youngest individuals (say, 0-4 years), a reduction in prevalence P is likely to be achieved largely by reducing the incidence of infection, thereby enlarging the pool of people never infected. Moreover, such young children appear biologically incapable of mounting a protective immune response like that of an adult chronically exposed to malarial infection" (see p. 16). In short, the fever cases and blood cases for the population under age 5 will become part of the never-infected group and for ages 5 and older will become part of the previously infected group.

So, for the 0 to 4 age group, the post-mcp never-infected subpopulation would consist of those who would be never infected even in the absence of an mcp and those who are never infected because of the programme:

$$p_0' = p_0 + s_F p_f + s_B p_b. \quad (7)$$

For those already born (ages 5 and over) it is assumed that those fever cases and currently infected cases will take on the mortality risk of the previously infected. This assumption is certainly more realistic under a stable malarial environment than under an unstable environment. In addition, those who do not get infected as a result of a programme are certainly different from those who do not get infected due to their own immune responses.

Therefore, with respect to the population aged 5 and over, the proportion of subpopulation of previously infected cases after the mcp would be

$$p_i' = p_i + s_F p_f + s_B p_b. \quad (8)$$

The success coefficients used for the proportions of the subpopulation need not be identical to those used for the death rates. Different activities of a multifaceted control programme may affect one success coefficient, other activities may affect more than one. But, as also indicated by Cohen in his numerical example, if there is no reason to do otherwise, it is expedient to assume all success coefficients are identical.

We now know the post-mcp proportions for the four groups, as well as the post-mcp death rates for the fever cases and currently infected cases (the previously infected and never-infected death rates without the exogenous changes remain unchanged). Post-mcp mortality can be derived from equations (1) and (2) and from replacing p and d values as shown in equations (3) to (8).

Eradication programmes. After an eradication programme, the subpopulations of the fever cases and currently infected cases will become part of the previously infected subpopulation, while new cohorts will be born into and remain in the never-infected group. Under eradication programmes, no new malaria cases occur and malaria is eventually eliminated (incidence is zero). Cohen points out that the "subpopulation [of previously infected cases] gradually loses members by death, and the excess mortality of individuals originally in [this group] gradually declines as any constitutional weakening and immunological stimulation of prior malarial infection fade into the past" (see p. 17). As mortality runs its course, the entire population will become never infected. Under an eradication programme, because the decline in the proportion of the previously infected individuals and in the excess death rate depends on the passage of time, the model incorporates the time factor.

The death rate after the mcp is a weighted average of those in the relevant subpopulations, just as with the chemotherapeutic and multifaceted programmes. However, after an eradication programme only the subpopulations of the previously infected and never infected are considered; because of eradication there are no more fever cases and no more currently infected cases. In addition to the time factor, age must be explicitly considered to separate those cohorts both before the mcp from those born afterwards. Simply then

$$d'(t, n) = p_0'(t, n)d_0'(t, n) + p_i'(t, n)d_i'(t, n) \quad (9)$$

where t refers to years since eradication and n refers to age group (n, n + 4).

The proportion of the population aged (n + 5, n + 9) at time (t + 5) that is never infected $[p_0'(t + 5, n + 5)]$ is calculated recursively through traditional cohort component projection techniques. The proportion previously infected is, of course, the complement:

$$p_i'(t + 5, n + 5) = 1 - p_0'(t + 5, n + 5).$$

Hence,

$$p_0'(t + 5, n + 5) = p_0'(t, n){}_5SR_t^0/[p_0'(t, n){}_5SR_t^0 + p_i'(t, n){}_5SR_t^i]$$

where ${}_5SR_t^0$ and ${}_5SR_t^i$ are the five-year survival ratios for the never-infected subpopulation and previously infected (immune) subpopulation, respectively. (In actuality, Cohen roughly approximates these survival ratios by the complement of the five-year death rate.) That is, the proportion of the never-infected cases at a given time for a given cohort is the cohort survivors in the group divided by the total population. The total population, as previ-

ously noted, consists of the sum of the never-infected cases and the previously infected cases.

The death rate of the never-infected subpopulation is unaffected by the eradication programme, so $d_0'(t, n)$ is identical to d_0 before the intervention, except for any exogenous change in mortality. The death rate of the previously infected subpopulation is assumed to decline from d_i at the time before eradication to d_0' sometime in the future, as the excess mortality due to the chronic malarial condition recedes. The rate of decline in excess mortality, which Cohen refers to as the decay function, must be specified by the user. Cohen suggests a linear or exponential decline function in the absence of any data to suggest otherwise.

Conclusion

Although this article has tried to explain the spirit and rationale of Cohen's model on the mortality effects of three types of malaria control programmes, it is nevertheless essential for the epidemiologist or demographer who chooses to apply the model to study carefully the Cohen paper where all elements are fully developed, and strengths, weaknesses and hidden complexities are explored in detail. We look forward to further applications of this model as necessary data are collected and tabulated.[2] We also hope that this model suggests an approach for estimating the expected mortality effects of different types of mortality control programmes for risks other than malaria.

Notes

[1] Cohen defines c as "the 'competing risk control coefficient'. The operational meaning of c is that competing risks reduce a chemotherapeutic programme's effective success so that the excess mortality in the population of fever cases is, on average, $d_f' - d_0' = (1 - cs_f)(d_f - d_0)$" (for details, see p. 15).

[2] Regrettably, when the model was developed, only data from the Garki project (Molineaux and Gramiccia, 1980; World Health Organization, 1975) were available.

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ESTIMATING THE EFFECTS OF SUCCESSFUL MALARIA CONTROL PROGRAMMES ON MORTALITY *

Joel E. Cohen **

SUMMARY

This paper proposes a new, empirically based model to measure the expected effects of successful malaria control programmes on human mortality levels and structure. In the model, the human population (which may be interpreted as a specific age group) is partitioned into four subpopulations: individuals never infected with malaria, individuals with fever from malaria, individuals currently infected with malaria but without fever, and individuals previously but not currently infected with malaria. Three kinds of malaria control programmes are considered: chemotherapeutic programmes (which attempt to reduce the mortality of fever cases); multifaceted programmes (which attempt to reduce both the prevalence of infection and fever, and the excess mortality of individuals with infection or fever); and eradication programmes (which attempt to bring the incidence and prevalence of malarial infection permanently to zero).

The model produces a range of estimated death rates. The range depends on uncertainty about the effective success of control measures. A numerical example, based on the data of the Garki project in northern Nigeria, is discussed. Some alternatives to the model are mentioned. Some strengths and weaknesses of the model are described and evaluated. And some recommendations are offered for the collection and analysis of data in the intersection of malarial epidemiology and demography.

INTRODUCTION: WHAT IS THE PROBLEM?

Large numbers of people are at risk of sickness and death from malaria. In 1986, the WHO Expert Committee on Malaria estimated that approximately 2.1 billion people live in "areas where a reduced level of [malarial] infection is maintained by the continued application of antimalarial measures" and another 370 million live in areas where malarial transmission continues without any organized efforts at control (World Health Organization, 1986, p. 9). Wyler (1983) gives a good general review of the situation of malaria and research on malaria.

The present paper, which describes an empirically based model to estimate the expected effects of successful malaria control programmes (mcps) on human mortality levels and structure, begins by reviewing previous empirical and theoretical studies of the impact on mortality of malaria and its control.[1] Empirical studies lead to three generalizations that a model must explain. The uses and limits of four theoretical approaches to measuring the mortality effects of successful mcps are identified.

An analytical portion of the paper suggests the elements that a model should relate: the human subpopulations that are differentially affected by malaria, and the kinds of mortality corresponding to each subpopulation; the major variants of mcps; and some major ecological and social variables.

The synthetic part of the paper integrates these elements in simple mathematical models that relate malarial epidemiology to human mortality. One model is illustrated by a numerical example based on the Garki project (Molineaux and Gramiccia, 1980). Finally, some strengths and weaknesses of the proposed models are described, some alternative approaches to modelling are sketched and some recommendations for action proposed.

The problem tackled in this paper is very difficult. Molineaux and Gramiccia (1980, p. 247) wrote that "Computation of the expected demographic consequences of malaria control could only be made with a relatively large error. Even so, it may be worth attempting with the limited data available." Ewbank and others (1986, p. 71) conceded frankly that "we are not even sure of the amount of mortality associated with malaria".

The difficulty arises in part, but not entirely, from limitations of empirical knowledge. For example, where malaria is suspected of causing many deaths, data on the causes of death are rarely satisfactory: usually, no medically trained individual observes a death and vital statistical systems may not provide reliable national data even when individual reports are correct. Moreover, if a physician, nurse or medical aide observes an acute illness

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caused by malaria, he or she is likely to interfere with the course of the illness. Though medical personnel may provide reliable data on the causes of those deaths that they observe, their very presence systematically alters the situation. The data they provide, though correct, may not characterize other parts of the country if the country is poor and has few medical personnel. Further, because the force and course of malaria depend on local environmental conditions, accurate information from one locality may not apply regionally or nationally. Finally, consider the difficulties of measuring the impact of malaria on mortality from an experimental mcp in the field, with well chosen and carefully observed treated and untreated populations. If malaria is a serious problem, it is likely that the untreated population will be able to buy antimalarial drugs and will, like the treated population, use them at least for acute episodes of fever, thereby diminishing the apparent impact of the mcp on mortality. On the other hand, if drugs or medical personnel are involved with the treated population, it is likely that they will reduce mortality due to other causes as well as malaria, thereby exaggerating the apparent impact of the mcp on mortality. Experimental field mcps, which might at first appear to be the ideal way to determine the impact of malaria on mortality, are fraught with difficulties of interpretation.

A deficiency as important as limited empirical knowledge is the lack of adequate theory. Inadequate theory limits the ability to organize available information and to guide the collection of additional data. Models could organize some of the available facts and indicate what additional facts would be most useful.

Empirical approaches to the problem

Molineaux (1985) reviewed comprehensively empirical knowledge of the impact on mortality of malaria and its control. Three of his major generalizations provide points of reference to be explained by the models that follow.

First, for a given prevalence of infection with the micro-organism that causes the most severe form of malaria (*Plasmodium falciparum*), the level of adult mortality from malaria depends critically on whether malaria in the given region is stable or unstable. Other factors, such as nutrition and concurrent infections, also affect adult mortality from malaria, but the local ecology of malaria is a dominant variable. Malaria transmission is said to be stable if transmission remains high year-round (though possibly with seasonal fluctuations) and high from year to year. Stable malaria is found, for example, in the Garki area of northern Nigeria (Molineaux and Gramiccia, 1980, p. 116). Malaria transmission is said to be unstable if transmission is essentially interrupted during at least one season of the year or fluctuates from a very low level to a very high level from year to year.

An area with sporadically recurrent epidemics of malaria, which strike unpredictably from year to year, has unstable malaria, but unstable malaria may also occur without epidemics. As Dietz pointed out (K. Dietz, personal communication, 30 December 1986), malarial transmission is interrupted for several months a year in some villages within the Garki area. It may be appropriate to treat those villages as having unstable malaria if they are isolated from the other villages, or as having stable malaria if there is enough migration among the villages for people to acquire malaria while out of their home village.

According to Molineaux (1985, p. 35), when the transmission of *P. falciparum* is stable and intense at a high level, adult mortality directly attributed to *P. falciparum* is probably very low (because the surviving adults acquire a clinical but not sterile immunity). By contrast, when the transmission of *P. falciparum* is unstable, then adult mortality directly attributed to *P. falciparum* may be very substantial during episodes of intense transmission, such as epidemics. In either case, intense transmission causes considerable mortality among infants and young children (ages 0-4).

Secondly, the mortality directly attributed to malaria may, under certain circumstances, be small compared to the enhancement that malaria causes in the mortality directly attributed to other causes. Molineaux (1985) cites Newman's (e.g., 1965, 1977) and Gray's (1974) analyses of mortality in Sri Lanka, which suggest that the indirect effects of malaria on mortality were two to four times as large as the direct effects; and Giglioli's (1972) analysis of mortality in Guyana, which suggests that *P. falciparum* increased mortality from respiratory disease there. According to this generalization, malarial control may be expected to reduce mortality directly attributed to malaria and should, perhaps with some lag, lead to a larger reduction in mortality due to indirect effects.

Thirdly, in apparent contradiction to the preceding generalization, malarial control that reduces mortality directly attributed to malaria may lead to a much smaller net reduction in overall mortality, because most of the individuals saved from death due to malaria rapidly die from another cause. This phenomenon is a reasonable interpretation of what was observed in Garki (Molineaux and Gramiccia, 1980). Similarly, in a Gambian village studied by McGregor (1964) and described by Molineaux (1985), the total number of deaths among infants and young children varied little from year to year, although the distribution of deaths by cause between malaria and measles varied greatly from year to year.[2]

The indirect action of malaria on mortality (Molineaux's second generalization) suggests that controlling malaria mortality would have a greatly amplified effect on overall mortality. The interaction of malaria with competing risks (Molineaux's third generalization) suggests that controlling malarial mortality would have a much smaller effect on overall mortality. How are these two generalizations to be reconciled?

One explanation, suggested by Preston (S. H. Preston, personal communication, 3 December 1986), is that both phenomena may be artifacts of poor reporting or coding of the cause of death. As he points out, if deaths actually caused by malaria were attributed statistically to another cause, then controlling malaria would lead to a reduction in mortality that had wrongly been attributed to other causes. If, on the contrary, deaths actually due to other

causes were attributed to malaria, then controlling malaria would lead to a smaller than expected reduction in deaths, as if competing risks were at work.

For analyses based on large numbers of cases assembled through the official system of vital statistics of a developing country, there is no denying that these sources of error may be at work. The relative magnitudes of these sources of error are likely to be unknown. When observed in small, special-purpose field studies, such as those of McGregor (1964) and Molineaux and Gramiccia (1980), Molineaux's second and third generalizations probably cannot be explained entirely as artifacts.

I accept these generalizations and go beyond the analysis of Molineaux to suggest tentatively the following. Where malaria control led to a reduction in mortality which was far greater than the reduction in mortality directly attributed to malaria, the country was undergoing a process of cultural, educational and economic development far more extensive than the efforts to control malaria. For example, in Sri Lanka, the crude death rate began to decline long before the post-Second World War antimalarial spraying campaign. It has been argued that the decline resulted from improved maternal and child welfare services, improved nutrition and improved medical services, among other causes (Meegama, 1986). By contrast, in the Garki research programme, the only interventions were mass drug administration and residual spraying, both directed primarily to malaria (Molineaux and Gramiccia, 1980). Other factors contributing to the population's high level of mortality were not directly altered.

This view of the findings of Molineaux (1985) leads to two conclusions. The first, also Molineaux's first, is that adult mortality directly attributed to *P. falciparum* is likely to be low where malarial transmission is stable, and is likely to be higher where malarial transmission is unstable. The second is based on a tentative reconciliation of two findings of Molineaux. In a setting of general development, the indirect mortality benefits of malarial control may be several times the reduction in mortality directly attributed to malaria. But when malarial control is carried out in isolation, the reduction in overall mortality may be considerably smaller than the reduction in mortality directly attributed to malaria.

As Singer pointed out, if this second conclusion is correct, it has a parallel in the conditions under which programmes intended to lower fertility are most effective (B. H. Singer, personal communication, 16 December 1986). Previous studies of the relation of socio-economic factors to mortality from malaria include those of Pampana (1955) and Banguero and others (1982). It would not be surprising if portions of the reality were more complicated than the two simple conclusions just offered.

Theoretical approaches to the problem

Several theoretical approaches to estimating the impact on mortality of malaria and its control have been proposed. The four reviewed here are the Bernoulli-Makeham procedure of demographers (e.g., Spiegelman, 1968), based on the assumption of independence among causes of death; Peterson's (1976) extension of this procedure to allow for dependence among causes of death; mathematical models of malarial transmission (e.g., Dietz, 1986); and regression analysis (e.g., Newman, 1977; Ewbank and others, 1986, p. 63).

The Bernoulli-Makeham procedure, reduced to its simplest form, classifies all deaths of individuals in a given age group into those deaths caused by malaria and those deaths with other causes. Some ambiguities in the concept of a death caused by malaria will be clarified later. The Bernoulli-Makeham procedure assumes that deaths caused by malaria are reduced by x per cent, through means left unspecified, and computes the overall reduction in deaths in the age group. The computation assumes further that the individuals who are saved from dying of malaria have a risk of death from other causes that is identical to that of all other individuals in the age group. Therefore, some of the individuals saved from dying of malaria will die of other causes, and the total number of deaths from other causes will rise accordingly. However, since not every individual saved from dying of malaria will die of other causes while in the age group, there will be a net decrease in the number of deaths in the age group. The age-specific death rate will decline by y per cent as a result of the x per cent decline in deaths due to malaria, where y is smaller than x.

Preston and others (1972) applied the Bernoulli-Makeham procedure to national statistics on broad classes of causes of death. They point out, as have others before and since, that the assumption of independence among causes, which underlies the method, cannot be tested using the data furnished in conventional tables of death by cause. The observations of Molineaux (1985) suggest that the assumption of independence between malaria and other causes is unlikely to be true. It follows that the Bernoulli-Makeham procedure, by itself, is not likely to be useful for estimating the mortality impact of successful mcps. The Bernoulli-Makeham procedure cannot accommodate the observation that in some cases the indirect reductions in mortality greatly exceed the reductions in mortality directly attributed to malaria. Neither can it accommodate the observation that in other cases competing risks (e.g., measles) completely counterbalance reductions in mortality directly attributed to malaria.

Peterson (1976) considered the extremes of possible dependence between two competing risks of death, which are "malaria" and "other" in the present context. At one extreme, if malaria and other causes are positively dependent, then every individual who would have died from malaria before control would die at the same age from another cause if death from malaria were prevented. Hence, under this extreme hypothesis, a "successful" mcp could result in no decrease in the age-specific death rate. This extreme hypothesis of positive dependence among causes represents precisely the compensation between malaria and measles that McGregor described in a Gambian village. For an mcp in isolation from economic and social development, the net mortality benefit of an mcp that succeeded in eliminating or reducing deaths from malaria could be as low as zero.[3]

At the other extreme, if malaria and other causes are negatively dependent, then every individual who would have died (in a given age interval) from malaria before control would not die (within the age interval) from any other cause after control. Under this extreme hypothesis, every death due to malaria that is postponed beyond the end of the current accounting period by a successful mcp would contribute to a reduction in the net death rate. This negative dependence among causes leads to complete survival, within the specified age interval, of individuals saved from dying directly of malaria. This hypothesis fails to represent the indirect effects of successful mcps described by Molineaux and others; it does not allow the elimination of malaria to lower the risks of death from other causes. It may be useful to compute the extreme reduction in death rates implied by Peterson's hypothesis of negative dependence among causes, but it will be important to recognize that still larger reductions are possible.

Both the Bernoulli-Makeham procedure and Peterson's extension to dependent causes take as given the number of deaths from malaria that are averted in each age class by a successful mcp. Where are these numbers to come from? Control programmes, successful or not, are usually specified by administrative and technical inputs and are monitored by epidemiological measures of coverage. It seems natural to expect that mathematical models of the transmission dynamics of malaria link the activities of an mcp with its achievements in reducing mortality from malaria. Unfortunately, the available models do not. Bailey's book (1982) on mathematical models of malaria indexed neither "mortality" nor "death rates". Dietz's (1986) comprehensive review and analysis concentrated mainly on a discussion of the relation between equilibrium prevalence of infection and vectorial capacity depending on simplistic assumptions about density-dependence in man and vector. No linkage with malaria-specific or general death rates was discussed. It therefore is necessary to build a linkage between malarial epidemiology and mortality on top of or in parallel with existing mathematical models of the transmission dynamics of malaria. A way to do this is suggested below.

Ewbank and others (1986) used regression to estimate the mortality consequences of successful mcps. For each province in Kenya, they used as the independent variable (X) the percentage of out-patient cases (in 1979) that were diagnosed as malaria. As the dependent variable (Y), they used the infant mortality rate per thousand. They then estimated a linear regression $E(Y) = a + bX$ for the average or expected infant mortality rate $E(Y)$ in a province with percentage of malaria X. When X is replaced by 0 in this equation, the expected infant mortality rate is just $E(Y) = a$. Ewbank and others (1986, p. 63) concluded: "This analysis suggests that in high mortality districts, eliminating malaria would reduce the infant mortality rate by 15 points. If an equal number of deaths were prevented at all other ages, malaria would be responsible for about 8 per cent of all deaths in districts with a high incidence of malaria."

The use of the word "suggests" is appropriate because there are well-known problems with using regression equations for prediction (e.g., Tufte, 1974, pp. 80-81). As Ewbank and others pointed out, it is doubtful whether the percentage of malaria cases diagnosed in out-patients bears any reliable relation to the prevalence or incidence of malaria in the population from which the infant mortality rate is measured. But suppose, in order to focus on other, more important questions, that an appropriate measure (X) of malarial incidence or prevalence and an appropriate measure (Y) of age-specific mortality could be defined and measured reliably. When Ewbank and others added to the regression equation variables representing ecological zones, the malaria index was no longer a significant predictor of infant mortality. Reducing malarial incidence or prevalence X to zero in reality, as opposed to the regression equation, might have no effect on infant mortality. On the other hand, if malarial incidence were reduced to zero by extensive programmes of environmental alteration, public health education and economic development (leading, for example, to screened windows), then the reduction in infant mortality might be far greater than that predicted by the regression equation.

Another problem with extrapolating from the given regression equation is that the observed percentage X of out-patient cases diagnosed as malaria ranged from 6 per cent to 30 per cent. It is notoriously unreliable to extrapolate regression equations outside the range of data for which they are estimated, in this case to $X = 0$, because there is no way of knowing whether X and Y are linearly related, or, if so, with the same values of a and b, outside the observed range of data.

Ewbank and others (1986, p. 63) were careful to point out reasons why the proportion of deaths caused by malaria might be much smaller or much larger than the 8 per cent they estimated. The conceptual difficulties described above, and many others, make regression equations a doubtful tool for estimating in advance the effects on mortality of successful mcps.

However, it is important to distinguish prediction from measurement. While the above arguments suggest that regression equations are an unreliable way to estimate the future reduction in mortality that would follow from a hypothetical mcp, there is nothing implausible about using regression equations to summarize observed effects of an mcp on mortality, as Newman (e.g., 1977) and Gray (1974) have done with Sri Lankan data.

In summary, the classical life-table analysis of Bernoulli and Makeham, its modern extension by Peterson to dependent competing risks, mathematical models of malaria transmission, and regression equations all suffer from serious defects as models to predict the impact of mcps on mortality. What follows is an attempt at a fresh analysis and some new models.

Analysis: control programmes, mortality, environment

How will a successful mcp affect the level and structure of mortality? In this question, the independent or input variable is "a successful mcp" and the dependent or output variable is "the level and structure of mortality". This analytical part of the paper specifies the meas-

ures of input, output and environment that a model needs to relate.

First, this paper will describe how the people affected by malaria are identified. It will then categorize deaths associated with the presence of malaria in a population. Then it will describe the possible forms of mcps and connect these forms with the deaths caused by malaria. Because given mcps have different effects on mortality, depending on the human and natural situation, it will also specify the environment in which the input and output variables interact.

Specifying the people affected by malaria

The aim of mcps is to protect people at risk of malaria. The aim of this paper is to estimate the reduction in mortality that will follow successful mcps. Both aims require operational means of identifying the people affected by malaria.

This section describes three major methods of identifying the people affected by malaria. For each method, the risk of false negatives (failing to identify a person who is affected by malaria) and the risk of false positives (identifying a person as affected by malaria who is not so affected) are described. On the basis of these methods, a partition of the malaria-affected population into four subpopulations is proposed: never infected, fever cases, currently infected (but not fever cases) and previously infected.

Parasitemic cases. A drop of blood is taken from a person and malarial parasites are identified by some standardized procedure. The procedure has traditionally been microscopic examination of the blood (Hunter and others, 1966) but DNA probes for the genetic material of the parasite are under development (e.g., Wirth and others, 1986). The variants on the microscopic and diagnostic techniques employed are not germane here. What will be considered here are diagnostic techniques that detect the presence of the parasite, rather than the immune reactions of the person.

Routine microscopic examination may miss some infections (in the Garki project, roughly 5 per cent to 25 per cent, depending on the species of malarial parasite (Molineaux and Gramiccia, 1980, pp. 112-114)), but it is unlikely to identify anyone without malaria as a malarial case. On balance, the reported number of parasitemic cases is likely to understate the number of individuals infected with malaria.

Where malaria is endemic, malarial parasites may appear in the blood of many people who consider themselves healthy. Thus, most "parasitemic cases" are not "fever cases", as defined next. However, malaria-infected individuals, even if they do not consider themselves sick from malaria, have a higher risk of death (World Health Organization, 1975) than they would have if they were not infected with malaria.

The fraction of people who are parasitemic cases will be called the parasite rate. The parasite rate may be crude (referring to an entire population in a defined area), age-specific or age-standardized. The parasite rate may refer to a particular species of *Plasmodium* (the genus of the various species of malarial parasites), to particular stages in the life cycle of one species (e.g., *P. falciparum* gametocytes), or to all malarial species.

Fever cases. Breman and Campbell (1986) propose that "a probable case of malaria is a person having fever (above 38°C, 100.4°F, if a thermometer is available) associated with the current illness. Chills, sweats or other signs and symptoms consistent with malaria may also occur but are not required for treatment. The person should live in or come from a malarious area, and there should be no other apparent cause of illness". A person meeting these criteria will be called a fever case.

Molineaux and Gramiccia (1980, p. 258) report that "screening the [Garki] population for temperatures of 37.5°C or more would have detected only 14.6% . . . of the [parasitological] positives below 9 years and only 3.4% . . . of the positives above 9 years of age. These findings are probably characteristic of a situation of intense transmission and relatively strong immunity of the survivors". On the other hand, fever cases probably include people who have no malarial infection but who do have fever from other causes, such as viral infections, that are not easily identified under field conditions. Molineaux and Gramiccia (1980, p. 144, figure 38) found that, in the Garki project, the most intensive malarial control efforts reduced the percentage of children less than 9 years old positive for *P. falciparum* in the wet season from approximately 80 per cent to approximately 5 per cent, while, at the time of the latter measurement, the prevalence of body temperatures of 37.5°C or more among children less than 9 years old was 11 per cent in an unprotected village and 4 per cent in the most intensively protected villages (Molineaux and Gramiccia, 1980, p. 256, table 28). The reduction in the prevalence of fevers was far less than the reduction in the prevalence of infection. Of course, it is possible that fever cases were atypically resistant to the efforts to control malaria.

On balance, the figures of Molineaux and Gramiccia suggest that there are many more parasitemic cases than fever cases under stable, intense malarial transmission.

The number of fever cases measures the acute burden of malarial disease perceived by the population. The criteria used to define fever cases may be workable in the field where microscopy is not possible. As a first approximation, it will be assumed that all fever cases are parasitemic cases. The fraction of people in a population who are fever cases will be called the fever rate.

Immune cases. Malarial infection challenges the immune system of people who survive long enough to develop a functioning immune system (e.g., Voller and others, 1980). Examination of the spleen and of the blood serum are the two main immunological approaches to determining whether an individual is affected by present or prior infection with malaria.

The spleen helps form antibody to antigens which, like malarial parasites, are particulate and distributed via blood (Humphrey and White, 1970, p. 263). Splenomegaly (enlargement of the spleen) often follows chronic malarial infection. Splenomegaly is determined clinically by palpating the spleen. Clinical scales exist for measuring the extent of enlargement. The spleen rate is the

prevalence of (fraction of people in a population having) a specified degree of splenomegaly. The population on which a spleen rate is based may be the entire population or a particular age group (Hunter and others, 1966, p. 337). The spleen rate has been widely used in malarial surveys to estimate the chronic intensity of malarial transmission in a population.

Individuals whose spleens have not yet enlarged in response to malarial infection, or whose spleens have enlarged too little to be palpable, or whose enlarged spleens have subsided to normal size, will not be numbered as having splenomegaly. Individuals without malarial infections whose spleens have enlarged in response to non-malarial (e.g., schistosomal or other helminthic) antigens will be numbered as having splenomegaly. The balance of false negatives and false positives, resulting from using splenomegaly as an indicator of chronic malarial infection, is not clear. However, it is consistent with past practice and reasonable until better indicators are available to suppose that spleen rates are positively associated with the prevalence of chronic malarial infection in malarial areas (cf. Newman, 1965, 1977).

Another principal approach to measuring the impact of malaria is via the blood serum. Many different immunological assays may be performed using serum. The assays produce a titre, that is, a measure of the concentration of a specified serum antibody. Commonly, one titre is chosen by the investigators as a threshold and individuals whose titre exceeds the threshold are considered positive for that assay.

Different assays measure different features of the immune response. To illustrate, the Garki project (Molineaux and Gramiccia, 1980, chap. 6) performed six assays: measurements of immunoglobulin G (IgG) and immunoglobulin M (IgM), a precipitin (Ouchterlony) test, two indirect fluorescent antibody (IFA) tests, and an indirect haemagglutination test. No relation was found between IgG and infection by the malarial parasite (as determined by microscopic examination of blood) nor, after the first few years of life, between the IFA test for *P. falciparum* and malarial infection. All but the IgG tests were positively associated with malarial infection among the young. "For 3 tests (IgM, precipitin and IHA) the relationship [to malarial infection] became negative in older children and adults, indicating that the test results were associated with protection" against infection (Molineaux and Gramiccia, 1980, p. 212). The three assays that are associated with protection in older children and adults also indicate the intensity of prior infection, since that infection induces the protective response. Finally, the IFA test for *P. malariae* was positively associated with infection throughout life. Other serological assays are under development (e.g., Wirth and others, 1986); their epidemiological characteristics remain to be determined.

If parasitemic cases are taken as the standard, the serological assays may produce false negatives because the threshold titre is set inappropriately high, because the reagents used in the assay are insensitive, or because the immune response is delayed relative to infection, that is, because a newly infected individual may not have had time to develop or display an immune response. The assays may produce false positives (relative to parasitemic cases) for diverse reasons: because a high IgM or IgG response is produced by some other infection; because the reagents for antimalarial antibodies cross-react with antibodies against other antigens; because the positive immune response measured by the assay is protective, having eliminated previous malarial infections and prevented reinfection; or because a non-protective immune response measured by the assay outlasts a malarial infection.

Notwithstanding the complexities of interpreting different serological assays, Molineaux and Gramiccia (1980, p. 207) concluded: "At the total population level, it is likely that all 6 tests used here are indicators both of contact with malaria and of partial immunity to it, that is, there is more malaria and a higher level of immunity in populations showing higher test results."

The serum rate of a population may be defined as the fraction of the population whose titre, on some specific test, exceeds a given threshold. The detailed interpretation of a serum rate depends on which test is used. The distribution of titres in a population tells more than the serum rate, but requires considerably more sophisticated analysis.

An immune case is defined as an individual who gives evidence of an immunological reaction to malarial infection. Such an individual may be said to be immunopositive (to malaria). As a first approximation, it will be assumed that every parasitemic case is an immune case.

In summary of this subsection, the size of the population affected by malaria may be described by the parasite rate, the fever rate, the spleen rate and several serum rates. These rates in fact describe different subpopulations.

On the basis of the biological facts just reviewed about malaria and its measurement, the population will be partitioned into four subpopulations. Individuals who are not fever, parasitemic or immune cases will be called "never infected" and the population in this category will be denoted p_0. The proportion of the population who are fever cases will be denoted p_f.[4] Individuals who are parasitemic cases but not fever cases will be called "currently infected" and the proportion of the population in this category will be denoted p_b. (The subscript b is a reminder that blood is drawn to diagnose parasitemia.) Finally, immune cases who are neither blood nor fever cases will be called "previously infected" and the proportion of the population in this category will be denoted p_i. Obviously, $p_0 + p_f + p_b + p_i = 1$. (The symbols introduced here [p_0, p_f, p_b, p_i] and in the remainder of this paper are listed and defined in the annex to the present article.)

In practice, although this partitioning of the population into four subpopulations could be done on the basis of a single cross-sectional survey, it would be preferable to estimate the point-prevalences of the four subpopulations from data collected using repeated observations of identified individuals over a period of time. The reason is that the fraction of individuals who are parasitemic (by

microscopic examination of blood slides) at a single observation appears to understate substantially the true prevalence of parasitemia in the population (e.g., Bruce-Chwatt, 1963; Molineaux and Gramiccia, 1980). Improved diagnostic procedures (e.g., Wirth and others, 1986) may alleviate this problem in the future.

Let P be the parasite rate and S the spleen or seropositive rate. Assuming that all fever cases are parasitemic cases, $P = p_f + p_b$; then p_b may be found from $p_b = P - p_f$. Similarly, assuming that all parasitemic cases are immunopositive, $S = P + p_i$, hence $p_i = S - P$. Under these assumptions or using direct measurement, the fractions p_0, p_f, p_b and p_i can in principle be estimated directly or indirectly from epidemiological measures commonly taken in malaria surveys.

Specifying mortality

In a population affected by malaria, people never infected with malaria have death rate d_0; fever cases have death rate d_f; currently infected people have death rate d_b; and people previously infected have death rate d_i. The death of an individual from each of these four subpopulations may be called, respectively, a death without malaria, a death from malaria, a death with malaria, and a death following malaria.

In principle, any death could be classified as one of these four kinds on the basis of information or material collected at or shortly before the time of death. In practice, in the developing countries where malaria remains a major problem, it is too much to hope that every death could be classified in this way, but it is not too much to hope that a carefully planned sample of deaths could be so classified to assist in planning and forecasting for an mcp. Hence, the four death rates are in principle measurable or estimable in conjunction with information about the relative sizes of the four subpopulations.[5]

If d is the death rate of the population, then

$$d = p_0 \cdot d_0 + p_f \cdot d_f + p_b \cdot d_b + p_i \cdot d_i. \qquad (1)$$

This equation may be used to check the consistency of the estimates of the fractions and death rates of the four subpopulations. The excess mortality of fever cases is $d_f - d_0$, of currently infected cases is $d_b - d_0$ and of previously infected cases is $d_i - d_0$.

All the above death rates and fractions refer to a population that can be pooled or specific, that is, heterogeneous or homogeneous, with respect to age or any other demographic characteristic. If the population contains all age groups, then the death rates are called crude rates; if a specific age group, then age-specific rates. If the population is a specific age group, the proportions p_0, p_f, p_b and p_i will obviously depend on the age. If the population consists entirely of individuals aged five years and older in an area where malaria is endemic and transmission is intense, it is reasonable, unless evidence to the contrary is available, to take $p_0 = 0$, since virtually everyone under such conditions has been infected with malaria.

In addition, malarial infection may be defined to include all species of malaria found in an area or any single species. From a biological perspective, it would be desirable to define malarial infection in terms of a single malarial species or of a specified combination of species, since the impact on mortality of an infection of *P. falciparum,* for example, differs from that of *P. vivax.*

However, the more homogeneous the population (in terms of age or malarial infection), the more detailed the demographic and epidemiological information required to measure or estimate the proportions and death rates of the subpopulations. When the model that follows is used for a real population, a want of data will quickly limit the possibility of using equation (1) for very narrowly defined populations.

Specifying malaria control programmes

After malaria eradication was abandoned in large parts of the world, the World Health Organization and the United States Agency for International Development outlined four approaches to malaria control, which are known as "tactical variants". The following presentation, based on Breman and Campbell (1986) and World Health Organization (1986), emphasizes the means that these tactical variants employ rather than their goals.

Tactical variant 1: chemotherapy. Sick people are treated with drugs. Attention focuses on children 0-4 years old who are acutely ill (and on pregnant women).

According to Breman and Campbell, this tactical variant "should be used in areas with high malaria prevalence, severe clinical illness and high case fatality rates, low socio-economic status, and limited experience in malaria programme administration. Most of rural Africa fits into this category." For convenience, an mcp following tactical variant 1 will be referred to as a chemotherapeutic programme.

Tactical variant 2: chemotheraphy, limited chemoprophylaxis and low-cost vector control. The acutely ill are treated with drugs. Drugs to prevent malaria are distributed to selected groups such as pregnant women. Vector control is initiated by voluntary measures such as mosquito netting.

Tactical variant 3: intensification and extension of tactical variant 2. In addition to all the activities of variant 2, a national or regional organization is used to apply more extensive vector control measures, and trained epidemiological personnel evaluate the actions taken and the results achieved.

An mcp based on tactical variant 2 or tactical variant 3 will be referred to as a multifaceted programme.

Tactical variant 4: eradication and surveillance. Expanded vector control operations are added to the activities of tactical variant 3, with a view to long-term eradication of malaria. Where malaria has been eradicated or is naturally absent, surveillance is undertaken to assure the continued absence of malaria. Asymptomatic carriers of malaria pose special problems for surveillance (Yekutiel, 1960); it may not be easy to decide when malaria has been eradicated from an area.

An mcp based on tactical variant 4 will be referred to as an eradication programme.

Recently, the WHO Expert Committee on Malaria reviewed the four tactical variants and suggested that

antimalarial programmes could be described more simply as using two major approaches: first, the treatment of people with malaria; and second, the long-term control of malaria transmission, in addition to the treatment of people with malaria. Both approaches are supposed to be carried out as part of the improvement in the general health services and control of the other major causes of preventable deaths (World Health Organization, 1986, pp. 48-49). What was defined here as a chemotherapeutic programme corresponds roughly to the first of these two approaches, while a multifaceted programme and an eradication programme correspond to different degrees of intensity of the second approach.

Various vaccines may eventually be available for use in mcps (e.g., Dame and others, 1984). However, as Halloran pointed out (M. E. Halloran, personal communication, 14 December 1986), vaccines are not currently available, it is not known when they will be available and their effects on mortality are unknown.

What constitutes a "successful mcp" (a term used often above and in what follows) requires definition. It would be convenient but useless to define a successful mcp as one that completely and permanently eliminates excess mortality in its target subpopulations (fever cases for chemotherapy programmes etc.). By this definition, there would be little uncertainty about the impact on mortality of successful mcps, but large uncertainty about whether an mcp should be considered successful. A less convenient but more useful definition would be that a successful mcp reaches completely and permanently its target subpopulations (so that, for example, all fever cases continually receive chemotherapy, whether or not chemotherapy saves their lives). Unfortunately, among real mcps, complete coverage is rare.

I propose that a successful mcp be defined as one that is both self-conscious and sustained. A self-conscious mcp has reliable procedures to measure the efforts it expends and the results (administrative, epidemiological and demographic) it attains. As Singer pointed out (B. H. Singer, personal communication, 1 May 1987), a self-conscious mcp also has reliable procedures to measure antimalarial activities other than the activities it initiates. For example, where chloroquine is already widely sold over the counter or by pedlars, an additional chemotherapy programme might have little effect on the mortality of fever cases, even though, in the absence of chloroquine, malaria was a major source of mortality. Proper evaluation of the impact of malaria on mortality requires a comprehensive picture of control activities. A sustained mcp adapts the control efforts applied to maintain malaria at some chosen level lower than that prior to the mcp; equivalently, it prevents a resurgence of malaria after a reduction is achieved. A sustained mcp need not constantly apply the same control measures, since the control measures appropriate to surveillance differ from those appropriate to an initial attack. By this definition, the Garki project (Molineaux and Gramiccia, 1980) was a successful mcp during its intervention phase. It was a successful research project, but not a successful control project, even after the intervention phase, as the transmission of malaria again increased.[6]

Connecting people affected by malaria with malaria control programmes

This section will attempt to link the tactics of mcps with the people affected by malaria. The proposed linkage is subject to revision by further information.

A chemotherapeutic programme is (by the above definition) directed to people who are acutely ill and to pregnant women. Therefore, apart from the pregnant women, a chemotherapeutic programme would presumably reduce malaria as a cause of death only among fever cases, since only they would be identified as acutely ill.

Chemotherapy might have diverse other effects, depending on whether drug treatment of an individual with malarial fever was or was not sustained over a long period and depending on whether the dosages applied were or were not sufficiently high to clear parasites from the treated individual. Taking account of both duration and dosage, there are four cases to consider.

Long-duration, high-dosage chemotherapy of an individual with a malarial fever could clear parasites. It would reduce substantially the p_b and p_i subpopulations in addition to the p_f subpopulation, because the treated individual would be prevented from becoming reinfected and would gradually lose his immunity. Nevertheless, other individuals might acquire infections without a period of fever and might shed those infections without either fever or drug treatment; thus, the p_b and p_i subpopulations would not necessarily vanish.

Long-duration, low-dosage chemotherapy would not clear parasites from treated individuals but would only lower the density of infection enough to remove fever. Such chemotherapy would convert individuals directly from fever to parasitemic cases.

Short-duration, high-dosage chemotherapy that cleared infections could convert fever cases to previously but not currently infected individuals, or could effectively convert fever cases to never-infected individuals by depriving them of the immunity resulting from prior malarial infections (Pringle and Avery-Jones, 1966). Such chemotherapy would provide only short-term protection against reinfection.

Finally, short-duration, low-dosage chemotherapy sufficient to interrupt a fever, as might be expected with self-administered drugs sold commercially, would temporarily convert fever cases to currently infected cases but would have little effect on the size of either subpopulation since nothing would prevent a recurrence of fever. This last case seems to be the most realistic description of actual chemotherapy programmes, unless they are part of a well-run research project.

This attempt at analysis suggests that chemotherapy could have multiple and non-obvious effects. The analysis could profit from more explicit and detailed modelling using a dynamic model of malarial infections, and should be considered subject to revision.

Individuals relieved of malarial infection by a multifaceted programme are still exposed to a (reduced) risk of reinfection. Individuals who had acquired protective immunity against malaria as a result of prior chronic

exposure to infection would not lose that protective immunity immediately. Hence, a multifaceted programme would be expected to have a much greater prompt effect on the parasite rate than on immunological indicators, for example, the spleen rate, or a serum rate associated with protective immunity, such as those (in the Garki study) based on IgM, precipitin and IHA assays.

The longer-term effect of a multifaceted mcp on protective immunity depends on how the programme works. For example, suppose the multifaceted programme lowered the parasite rate to 25 per cent of its former level and held the parasite rate steady at that level. If only one quarter of the population were infected at any one time but all individuals were infected at some time, then it is possible that no one would lose his or her protective immunity and the serum rates (based on assays for protective immunity) might remain constant. On the other hand, if the multifaceted programme really consisted of malarial eradication in areas where three quarters of the population lived and no control at all where one quarter of the population lived, then the serum rate of the protected three quarters would eventually drop to zero, while the serum rate of the unprotected quarter would not change. Under the latter hypothesis, because malaria control meant eradication in one region and no control in another, the serum rate would mirror the parasite rate. I will assume henceforth that major geographical differences in control would be identified and the effect of control on mortality in different regions would be analysed separately. I assume that partial control, that is, control short of eradication, will eliminate neither chronic exposure to malarial infection nor the possibility of maintaining a partially protective immunity.

A successful eradication programme would affect fever cases, parasitemic cases and immune cases, though the effects might differ depending on whether malaria were stable or unstable. For example, following eradication of stable malaria, previously infected individuals would, after some time, lose the immunity (and the sometimes pathological auto-immunity) produced by the antigenic stimulation of a chronic malarial infection. Thus, a successful eradication programme would eliminate the excess mortality associated with splenomegaly or malaria-induced auto-immune diseases, as well as any other mortality resulting from stable malaria.

Specifying the environment

As a first approximation, the environmental variables that modulate the effect of mcps on mortality may be classified as ecological and social.

The primary ecological variable that influences the effect of mcps on mortality is whether malaria is stable or unstable. First, suppose malaria is stable. Parasitemic cases old enough to have developed an immune response and to have survived many attacks of malaria (say 5 years and older) would be expected to have a small excess mortality $d_b - d_0$, relative to a situation of unstable malaria. But the excess mortality $d_i - d_0$ of previously infected individuals might be large, relative to a situation of unstable malaria, because of the weakening and auto-immunity caused by chronic malarial infection with *P. falciparum*. (The likely pathogenic effect of chronic *P. malariae* infection, for example, would be much less.) A plausible upper bound on the excess mortality $d_i - d_0$ is $d_f - d_0$. Now suppose malaria is unstable. The excess mortality of parasitemic cases $d_b - d_0$ may be quite substantial, especially at the older ages, while the excess mortality $d_i - d_0$ of previously infected individuals may be relatively small, because such individuals have not suffered chronic malarial infections. It is suggested below that this difference between stable and unstable malaria may lead to different patterns of falling mortality after malaria control.

The primary social variable modulating the effect of mcps on mortality appears to be whether the mcp is a single-purpose, isolated control programme (which may be a research programme, a demonstration programme or a single-purpose public health programme) or is part of an ongoing and comprehensive process that includes education, environmental and sanitary improvement, primary health care infrastructure and economic development. Where the mcp is isolated, it appears that, especially at the youngest ages, lives saved from malaria are often lost to other risks of death. (The same phenomenon has been demonstrated for lives of children vaccinated against measles in Zaire (Kasongo Project Team, 1981).) Where the mcp is part of extensive social development, it appears that the maximum possible improvements in mortality from mcps are realized. The difference between isolated mcps and mcps that are part of social development will be reflected in the value of a parameter that describes the action of competing risks in the models to be presented next.

SYNTHESIS: EFFECTS OF MALARIA CONTROL PROGRAMMES ON MORTALITY

Equation (1) expresses the death rate of a population as the mean of the death rates of four subpopulations, weighted by the relative sizes of those subpopulations. As already mentioned, for an analysis of age-specific death rates, equation (1) may be applied separately to different age groups.

Equation (1) focuses attention on how different kinds of mcps will affect the death rates and relative sizes of each of the four subpopulations. The models proposed now express the variables on the right side of equation (1) as functions of variables that describe the success of mcps and of ecological and social variables.

There is considerable uncertainty in both the structure of the models and the values of the parameters. Given the models' structure, the uncertainty in the parameter values implies a range of estimates of the death rate following mcps. This range of estimates has no meaningful interpretation as a confidence interval. However, if the models prove useful, it may be of interest, as Nedelman pointed out (J. Nedelman, personal communication, 3 December 1986), to assign subjective probability distributions to the parameters and to compute subjective distributions for the predicted death rate.

Let d' denote the death rate of the population after the implementation of an mcp. Throughout, the prime $'$ dis-

tinguishes a quantity after an mcp from the same quantity before an mcp.

Chemotherapeutic programmes

A chemotherapeutic programme affects the excess mortality, and could affect the size, of the subpopulation of fever cases.

Chemotherapy averts the death of a fever case, except where the malarial parasite is intractably drug-resistant or where the fever is unrelated to malaria. A chemotherapeutic programme that treats successfully a fraction s_f of the fever cases will be said to have a "success coefficient" s_f, which is a fraction between 0 and 1. It may be useful to think of s_f as the product of the coverage of the programme times the effectiveness of the programme in treating those individuals included in it. Assuming that all the excess mortality of fever cases is from malaria, a chemotherapeutic programme that effectively treats a fraction s_f of the fever cases could lower the excess mortality in the population of fever cases from $d_f - d_0$ to $d_f' - d_0' = (1 - s_f)(d_f - d_0)$. (I do not assume $d_0' = d_0$: the death rate of the never infected may change for exogenous reasons.)

However, competing risks of death may kill those fever cases saved from dying of malaria. To describe the action of competing risks, define c as the "competing risk control coefficient". The operational meaning of c is that competing risks reduce a chemotherapeutic programme's effective success so that the excess mortality in the population of fever cases is, on average,

$$d_f' - d_0' = (1 - c \cdot s_f)(d_f - d_0).$$

One may think of $c \cdot s_f$ as the effective success, in terms of mortality, of a chemotherapeutic programme that has apparent epidemiological success s_f. That is, the chemotherapeutic programme reaches a fraction s_f of fever cases and averts their deaths from malaria. But competing risks take away some of those lives saved, as if the programme reached only a fraction $c \cdot s_f$ of the fever cases in the absence of other causes of excess mortality.

For example, if all fever cases, whose death from malaria is averted by chemotherapy, die immediately from another cause, then competing risks are not controlled at all, and by definition $c = 0$. If all fever cases, whose death from malaria is averted by chemotherapy, die not immediately but within the year (or other unit of accounting) from another cause, then c would be small but strictly positive so that the death rate of the fever subpopulation would correctly reflect the person-years lived. A value of c between 0 and 1 means that fever cases saved from dying of malaria have some excess mortality, but less than untreated fever cases. At the other extreme, if fever cases, whose death from malaria is averted by chemotherapy, have only the risks of death of individuals never infected by malaria, then by definition $c = 1$ and competing risks are completely controlled.

In principle, c could be measured by longitudinal observations of treated fever cases. However, such a special-purpose study is likely to be impractical. It is still possible to estimate c crudely because, as noted, the action of competing risks appears to depend on the social context of the mcp. Where a chemotherapeutic programme is an isolated effort, it would be reasonable to make c small. Where a chemotherapeutic programme is part of a multifaceted or eradication programme, and these in turn are part of extensive social development, it would be reasonable to estimate c large, and close to 1. In any event, it would be desirable to compute d' twice, by considering the extreme cases $c = 0$ and $c = 1$, to see whether the uncertainty about c materially affects the estimate d'.

Another possible form of sensitivity analysis would be to consider other formulations of the success coefficient. The above formulation of the success coefficient and of effective success is additive because excess mortality is measured by the difference $d_f - d_0$. If excess mortality were measured by the mortality ratio d_f/d_0, a multiplicative version of the success coefficient could be defined, as pointed out by Horiuchi (S. Horiuchi, personal communication, 19 December 1986). For the extreme values 0 and 1 of the effective success coefficient, the results of using the additive and multiplicative versions are identical. The additive version is used here because the formula for it is simpler.

Chemotherapy prevents reinfection of the subpopulation of fever cases, only for a negligible period of time. During the period when a treated individual has no or few parasites in his peripheral blood, he has a reduced risk of infecting a mosquito that bites him, and that mosquito therefore has a reduced risk of infecting another human on which it feeds later. Thus, there is some impact, in principle, of chemotherapy on the subsequent incidence of human malarial infection. However, unless the chemotherapy programme reaches most of the population at risk nearly simultaneously, the impact of chemotherapy on subsequent malarial incidence seems likely to be small. In any event, it is virtually unmeasurable as a separate effect. At the same time, chemotherapy of fever cases does not diminish the flux of parasitemic cases into the subpopulation of fever cases. Hence, I suppose, as a first approximation, that in areas of intense transmission a chemotherapeutic programme has no effect on the size of the subpopulation of fever cases.

In summary, under a chemotherapeutic programme, the death rate and relative size of the subpopulation of fever cases are

$$d_f' = d_0' + (1 - c \cdot s_f)(d_f - d_0),$$

$$p_f' = p_f.$$

The mortality d_0' among never-infected individuals after the programme may differ from that d_0 before the programme for reasons unconnected with the mcp. Then the death rate d' of the population after the programme is in place, continually treating fever cases, is predicted to become

$$d' = p_0 \cdot d_0' + p_f \cdot d_f' + p_b \cdot d_b' + p_i \cdot d_i', \qquad (2)$$

with d_f' given above. Where no information to the contrary is available, it is parsimonious to assume that $d_0 = d_0'$, $d_b = d_b'$ and $d_i = d_i'$.

Though age is not mentioned in this model, the model is intended to be applied separately to age groups that have notably different values of the death rates or subpopulation proportions. As suggested by Ewbank (D. C. Ewbank, personal communication, 10 December 1986), suppose, for example, that if an attack of malaria coincides with weanling diarrhoea, the child has a high risk of death from or with malaria, but if the child suffers a malarial infection after the weanling diarrhoea is past, the mortality from or with malaria is much reduced. To anticipate the impact of a chemotherapeutic programme on children in this situation, it would be appropriate to apply the above model separately to pre- and post-weaning age groups, in combination with standard demographic cohort projection. Chemotherapy in the pre-weaning group would leave more children alive to benefit from the lower malarial death rates of the post-weaning group.

Multifaceted programmes

The success of a multifaceted programme may be measured by four average "success coefficients": s_f, the fraction of excess mortality from malaria that is prevented by chemotherapy among fever cases (exactly as in a chemotherapeutic programme),

$$s_f = 1 - [d_f' - d_0']/[d_f - d_0];$$

s_b, the fraction of excess mortality with malaria that is prevented by chemotherapy and chemoprophylaxis among currently infected cases,

$$s_b = 1 - [d_b' - d_0']/[d_b - d_0];$$

s_F, the fractional reduction in the size of the subpopulation of fever cases,

$$s_F = 1 - p_f'/p_f;$$

and s_B, the fractional reduction in the size of the subpopulation of currently infected cases,

$$s_B = 1 - p_b'/p_b.$$

How are these success coefficients to be estimated, short of carrying out the mcp and waiting to see what happens? The fractional reductions in excess mortality s_f and s_b could be estimated crudely from the mcp plan as the programme's anticipated coverage of the subpopulations of, respectively, fever cases and currently infected cases. The fractional reductions in the subpopulations of fever and currently infected cases could be measured after the fact, in the course of epidemiological surveillance once the mcp is in place. Alternatively, mathematical models of malarial transmission (see, e.g., Dietz, 1986; Gonzalez-Guzman, 1980) could be used in advance to derive a predicted post-control parasite rate P'; a "success coefficient" s_P for reducing the parasite rate could then be estimated from

$$s_P = 1 - P'/P$$

and, until better information is available, one could take $s_F = s_B = s_P$. The model presented here does not provide a prediction of the size of the subpopulations of fever and currently infected cases after a multifaceted programme is in place.

The people who leave the subpopulations of fever cases and currently infected cases as a result of multifaceted control may be allocated, in this model, to either of two subpopulations: those never infected, or those previously infected. Although age has not entered the model explicitly up to this point, it seems necessary to introduce age here.

For the youngest individuals (say, 0-4 years), a reduction in prevalence P is likely to be achieved largely by reducing the incidence of infection, thereby enlarging the pool of people never infected. Moreover, such young children appear biologically incapable of mounting a protective immune response like that of an adult chronically exposed to malarial infection. So for never-infected children, the size of the subpopulation changes from p_0, before multifaceted control, to

$$p_0' = p_0 + s_F \cdot p_f + s_B \cdot p_b \qquad \text{(0-4 years only)}$$

after multifaceted control.

For older individuals, the opposite allocation seems more sensible. Assuming the population is geographically homogeneous (or, if not, redefining the population so that it becomes geographically homogeneous), I will assume that everyone who would have been infected by malaria before control becomes infected with some steady mean frequency after control, and therefore maintains some immune response. This assumption is most plausible in an environment of stable malaria with fairly intense transmission, where older individuals experience chronic malarial infection. When the steady mean frequency of malarial infection is low, as in an environment of unstable malaria, I am assuming that there is at least some immunological memory of prior malarial infection among older individuals.

People who lose a malarial infection as a result of a multifaceted programme may be quite different from people who mount a protective immune response on their own. But it seems more reasonable that the excess mortality of formerly infected individuals saved from infection by a multifaceted programme would resemble the excess mortality of immune cases than that the formerly infected individuals saved from infection by a multifaceted programme would have no excess mortality at all, like the individuals never infected with malaria.

On the basis of these arguments and assumptions, among individuals aged five years and older, the relative size of the subpopulation of previously infected cases changes from p_i, before multifaceted control, to

$$p_i' = p_i + s_F \cdot p_f + s_B \cdot p_b \qquad \text{(5 years and older only)}$$

after multifaceted control.

There is no simple relation between the activities of a multifaceted control programme and the success

coefficients s_f, s_b, s_F and s_B. Indeed, the effects may be counterintuitive. Some activities affect only one measure; for example, chemotherapy of fever cases probably affects mainly s_f. Other activities affect more than one measure. For example, measures directed against the mosquito population, such as larviciding and altering breeding habitats; measures aimed at separating the mosquitoes from people, such as using mosquito nets, screening windows and spraying the insides of homes with residual insecticides; and chemoprophylaxis, aimed at preventing parasites from establishing the asexual life cycle in the human host, diminish the incidence, and thereby the prevalence, of malarial infection. But the measures aimed at the mosquito population and its attacks on people also diminish the frequency of superinfection (repeated inoculation of malarial parasites into individuals already infected with malaria).

In a population with endemic malaria, it is plausible that a reduction in superinfection would lower the mean interval between periods of being free of malaria, that is, would reduce the mean duration of a spell of malarial infection, which would in turn lower the excess mortality with malaria, especially among infants and children, "if chronic malaria affects adversely the general underlying condition of persons, which is likely" (Molineaux and Gramiccia, 1980, p. 246). The point is that an activity such as vector control or screening, which might appear aimed at incidence or at prevalence, may also affect excess mortality. Still other activities, such as mass drug administration, especially at a sufficiently high frequency, must affect all measures of success: s_f, by curing fever cases who would otherwise die; s_b, by reducing the density of parasites and the consequent pathology; and s_F and s_B, by terminating bouts of infection.

With currently infected cases as with fever cases, competing risks of death may reduce the demographic net effect of success in averting both deaths from malaria and deaths with malaria. The death rates in these two subpopulations become, after multifaceted control,

$$d_f' = d_0' + (1 - c \cdot s_f)(d_f - d_0),$$

$$d_b' = d_0' + (1 - c \cdot s_b)(d_b - d_0).$$

In summary, using the preceding steps, the death rate after multifaceted control is estimated to be

$$d' = [p_0 + s_B \cdot p_b + s_F \cdot p_f]d_0' + (1 - s_F)p_f d_f' \quad (3a)$$

$$+ (1 - s_B)p_b d_b' + p_i' \cdot d_i' \quad \text{(0-4 years only)}$$

$$d' = p_0' \cdot d_0' + (1 - s_F)p_f d_f' + (1 - s_B)p_b d_b' \quad (3b)$$

$$+ [p_i + s_B \cdot p_b + s_F \cdot p_f]d_i' \text{(5 years and older only)}$$

where d_f' and d_b' are given in the previous equation. Assume $d_0' = d_0$ and $d_i' = d_i$ unless there is information to the contrary. The logical requirement that the proportions of the four subpopulations sum to 1 both before and after control implies that $p_i' = p_i$ in (3a) and $p_0' = p_0$ in (3b). But if facts show that after multifaceted control individuals aged 0-4 years have been previously exposed or individuals aged 5 years and older have never been exposed, the simplifying assumptions made in constructing (3a) and (3b) should be modified accordingly.

A high death rate would be estimated by setting $c = 0$, so that competing risks neutralize any successes in reducing mortality from or with malaria. A low death rate would be estimated by setting $c = 1$, so that individuals reached by the programme attain the mortal risks of individuals never infected with malaria.

The same notation c is used for competing risks affecting both the p_f and the p_b subpopulations (as well as for all age groups, if age groups are to be considered) not because competing risks will have the same effect in all cases but because it is highly unlikely that data will be available that would justify assigning different values to c in different subpopulations. In the happy event that such data are available, different values of c could, of course, be introduced.

Eradication programmes

Like multifaceted programmes, eradication programmes lower the parasite rate. However, by reducing incidence to 0, eradication programmes, unlike control programmes, eventually eliminate the subpopulation of people previously infected. At the older ages, as time passes, the decline in the p_f and p_b subpopulations will be compensated by a rise in the P_0 subpopulation (never infected), rather than in the p_i subpopulation (previously infected).

The reason for the difference is that in a control programme, with a positive steady-state prevalence of malarial infection, individuals shuttle irregularly between a state of being infected and a state of being previously but not currently infected. The proportion infected is $P = p_f + p_b$ and the proportion previously but not currently infected is p_i. Individuals in the subpopulation p_i suffer the after-effects of sporadic (unstable) or chronic (stable) malaria. Under malarial eradication, the subpopulation p_i gradually loses members by death, and the excess mortality of individuals originally in p_i gradually declines as any constitutional weakening and immunological stimulation of prior malarial infection fade into the past.

Unfortunately, I do not know of quantitative information about the post-eradication rate of decline or asymptote of the excess mortality of previously infected individuals. As Singer pointed out (B. H. Singer, personal communication, 16 December 1986), it may be possible to extract generalizations about the post-eradication decline of excess mortality of previously infected individuals from the numbers of cases of malaria and numbers of malaria deaths in European countries (Bruce-Chwatt and de Zulueta, 1980). The difficulties of interpreting the available numbers are serious (for example, are the reported malarial deaths from, with or following malaria?) but the possibility of extracting useful information from them deserves study.

Because the decline in the subpopulation of previously infected people depends on the passage of time, it is natural to embed this model for the mortality effects of successful mcps in a standard demographic cohort-

component projection (e.g., Keyfitz, 1968). For concreteness, suppose the entire human population is treated as a collection of age-specific populations of individuals aged 0-4 years, 5-9 years and so on. These age groupings are conventional in demography.

Let $t = 0$ be the year eradication is achieved, so that $t \geqslant 0$ is the number of years since the last endogenous malaria case. Also, let (t, n) refer to the population of individuals aged n to $n + 4$ years in year t. Eradication means that there are no more fever cases and no more currently infected individuals, that is, for $t \geqslant 0$, $p_f'(t, n) = p_b'(t, n) = 0$, for $n = 0, 5, 10, 15, \ldots$.

It remains only to describe $p_0'(t, n)$ and $p_i'(t, n)$. For $t > n + 4$, the age group n to $n + 4$ contains only individuals born after eradication, and such individuals have never been infected, by the definition of eradication (and ignoring migration from other regions where malaria may still be endemic). Hence,

$$d'(t, n) = d_0'(t, n),$$

when $t > n + 4$. In this equation, the death rate of individuals aged n to $n + 4$ who were never infected with malaria is denoted as a function of time $d_0'(t, n)$ to allow for the possibility that this death rate may change as a result of factors having little to do with malaria or its control.

At $t = 5m$ years post-eradication ($m = 0, 1, 2, \ldots$), where $t \leqslant n$, the age group n to $n + 4$ contains individuals who were alive at the time of eradication and who were aged $n - t$ to $n - t + 4$ at that time. Let $p_0(0, n - t)$ denote the proportion of never-infected individuals in the age group $n - t$ to $n - t + 4$ just prior to eradication; similarly for $p_f(0, n - t)$, $p_b(0, n - t)$ and $p_i(0, n - t)$. Let $p_0'(t, n)$ and $p_i'(t, n)$ denote the proportions of never-infected and of previously infected individuals t years after eradication (when $t = 0$, this means just after eradication) in the age group n to $n + 4$. As stated above,

$$p_f'(0, n) = p_b'(0, n) = 0.$$

Immediately after eradication,

$$p_i'(0, n) = p_i(0, n) + p_f(0, n) + p_b(0, n),$$

for every n,

because eradication does not remove the fact of past malarial infection from the p_i subpopulation, and after eradication the fever and parasitemic cases join the ranks of the previously infected. After $n + 5$ years have passed since eradication, the population aged n to $n + 4$ years will have no previously infected individuals, because all individuals aged n to $n + 4$ will have been born after eradication. In symbols,

$$p_i'(n + 5, n) = 0, \quad n = 0, 5, 10, \cdots$$

Consequently, the excess mortality of the p_i subpopulation becomes 0 by $n + 5$ years post-eradication, because the p_i subpopulation vanishes.

What is uncertain, as I stated before, is how the aggregate excess mortality of the p_i subpopulation declines from $p_i(0, n)[d_i(0, n) - d_0(0, n)]$ just before eradication to 0 after $n + 5$ years. This ignorance will be described quantitatively, dealing first with the decline in per capita excess mortality $d_i'(t, n) - d_0'(t, n)$ and then with the decline in the size $p_i'(t, n)$ of the p_i subpopulation.

Just prior to eradication, $d_i(0, n) - d_0(0, n)$ is the average per capita excess mortality of previously infected individuals aged n to $n + 4$ years. After t years, this same cohort, now aged $n + t$ to $n + t - 4$, will, by definition, have per capita excess mortality

$$d_i'(t, n + t) - d_0'(t, n + t) = [d_i(0, n) - d_0(0, n)]f(t, n)$$

where the unknown function $f(t, n)$ describes the decay in excess mortality: $f(0, n) = 1$ and $f(t, n) \geqslant 0$ for all t and $f(t, n)$ is non-increasing with t. For practical applications of the model, it might be reasonable to assume that $f(t, n)$ declines exponentially and independently of the initial age n, for example, $f(t, n) = \exp(-Dt)$. Under this assumption, $(\ln 2)/D$ is the half-life of excess mortality of previously infected individuals, that is, the number of years required for the excess mortality of previously infected individuals to fall by half.

Two bits of evidence may be indirectly relevant to estimating the half-life of excess mortality of previously infected individuals. First, the half-life of the immunoglobulin IgG is estimated at 23 days, and other immunoglobulin classes (IgA, IgD, IgE and IgM) break down much more rapidly (Davis and others, 1973, p. 483). Secondly, schoolchildren living in a highly malarious area of East Africa who were protected from infection for between one and two months by the administration of antimalarial drugs displayed a substantial loss of clinical immunity to malaria when drug treatment was terminated, as many of the new infections caused more severe clinical symptoms and parasitemia than had prevailed before treatment (Pringle and Avery-Jones, 1966). The concordance between the half-life of IgG and the decay-time of clinical immunity to malaria is remarkable. Unfortunately, a direct link between these two facts and the decay of excess mortality of previously infected individuals remains to be made.

If t were measured in years, and excess mortality decayed exponentially with the same half-life as IgG, then $D = 11$ and the excess mortality of the previously infected would be unmeasurably small even half a year after eradication. The form of this decay may depend on the eradication programme; for example, medical care for previously infected individuals could accelerate the decline of their excess mortality. In the absence of directly relevant data, other functional forms and parameter estimates for the decline function $f(t, n)$ are equally plausible.

The decline in the size of the p_i subpopulation can now be described by simple equations. At time t, where $0 \leqslant t \leqslant n + 5$, the population aged n to $n + 4$ years

contains a fraction $p_0'(t, n)$ of individuals never infected, who suffer per capita mortality $d_0'(t, n)$, and a fraction $p_i'(t, n) = 1 - p_0'(t, n)$ of individuals previously infected, who suffer per capita mortality

$$d_i'(t, n) = d_0'(t, n) + [d_i(0, n - t) - d_0(0, n - t)]f(t, n - t).$$

As a rough approximation (better approximations are given by Keyfitz, 1968), using five-year death rates, and ignoring immigration and emigration, the number of never-infected individuals aged $n + 5$ to $n + 9$ y at time $t + 5$ is proportional to $p_0'(t, n)[1 - d_0'(t, n)]$ and the number of previously infected individuals aged $n + 5$ to $n + 9$ at time $t + 5$ is proportional to $p_i'(t, n)[1 - d_i'(t, n)]$. Consequently, the proportion of never-infected individuals aged $n + 5$ to $n + 9$ at time $t + 5$ is

$$p_0'(t + 5, n + 5) = p_0'(t, n)[1 - d_0'(t, n)] / [p_0'(t, n)1 - d_0'(t, n) + p_i'(t, n)1 - d_i'(t, n)].$$

The proportion of previously infected individuals is then

$$p_i'(t + 5, n + 5) = 1 - p_0'(t + 5, n + 5).$$

From these last two equations, given some assumed form for $f(t, n)$, the proportions $p_0'(t, n)$ and $p_i'(t, n)$ can be calculated for all times t post-eradication and all age groups n to $n + 4$. The crude death rate $d'(t, n)$ of the age group can be calculated as a weighted mean,

$$d'(t, n) = p_0'(t, n)d_0'(t, n) + p_i'(t, n)d_i'(t, n),$$

and the cohort can be projected by the standard cohort-component method.

The method depends, of course, on facts or assumptions about the decay function $f(t, n)$. Regardless of uncertainty about the form of $f(t, n)$, the end points at $f(0, n) = 1$ and $p_i'(n + 5, n) = 0$ are clear.

This very simple model can explain how the mortality gains from eradication can substantially exceed the direct reduction in mortality from or with malaria, as Molineaux (1985) observed. Ignoring age structure for a moment, and assuming that the background death rate d_0 of individuals never infected with malaria is constant before and after control, the aggregate post-control reduction in mortality, after all previously infected individuals have died, is $p_f(d_f - d_0) + p_b(d_b - d_0) + p_i(d_i - d_0)$. This reduction may greatly exceed the sum of the averted deaths from malaria $p_f(d_f - d_0)$ and averted deaths with malaria $p_b(d_b - d_0)$. The model not only explains this effect qualitatively, but shows that the size of the effect should depend on the time since eradication (cf. Giglioli, 1972). The model gives a way of estimating the size of the effect quantitatively, given facts or assumptions about the decay $f(t, n)$ of excess mortality of individuals previously infected.

It is plausible that an eradication programme can be put in place and made to hold only amid considerable social development. If so, it is equally plausible to evaluate the competing risk control coefficient c as $c = 1$, pending data to the contrary (particularly evidence of drug resistance).

Will an eradication programme cause a larger eventual decline in mortality where malaria is intense and stable or where malaria is epidemic and unstable? The answer depends on the relative size of the excess mortality in the p_i subpopulation versus that in the p_f and p_b subpopulations. Recall that the excess mortality of the p_i subpopulation is likely to be substantial in stable malaria compared with that in unstable or epidemic malaria. By contrast, the excess mortality of the p_f and p_b subpopulations is likely to be substantial in unstable or epidemic malaria compared with that in stable malaria. Eradication eventually eliminates both the p_i subpopulation and the $p_f + p_b$ subpopulations, and the magnitude of the population's mortality decline depends on the balance of the sizes of these subpopulations and the excess mortalities in them.

Numerical example: the Garki project

One of the most carefully planned and reported field experiments in malaria control is the Garki project (Molineaux and Gramiccia, 1980). The project was carried out in Kano State, northern Nigeria, by the World Health Organization in co-operation with the Government of Nigeria. Malarial transmission in this area is intense and stable: prior to any intervention, the crude parasite rate for *P. falciparum* infection for all villages combined exceeded nearly 50 per cent year-round, while the crude parasite rate for *P. malariae* infection exceeded 10 per cent year-round. Because eradication was neither a goal nor a result of the project, the Garki project is an example of a multifaceted programme. No effort was made to provide comprehensive social and economic development.

In the baseline phase of the project, the parasitology, immunology, clinical signs and demography of the study population were observed. In the intervention phase, several malaria control strategies, including mass drug administration and spraying of residual insecticides, were carried out in different combinations in different villages. Data from the villages that received the most intense treatment (follow-up units 5 and 7) will be the subject of attention here. During the intervention phase, some villages also received no treatment (follow-up units 1 and 2).

On the basis of differences in immune function and in the prevalence of infection with *P. falciparum* malaria, three age groups will be treated as separate populations: 0-4 years, 5-18 years and 19 years and older. Table 1 shows d, the death rates (from all causes) of these three age groups before control, and d', the corresponding estimates after control (estimated from the statistics on follow-up units 5 and 7). The post-control death rates (d') are uniformly and substantially lower than those

TABLE 1. DEATH RATES OF MALARIA SUBPOPULATIONS IN THE GARKI PROJECT

Line	Parameter	Population (age group in years) 0-4	5-18	19+
	Villages where treatments were applied (follow-up units 5, 7)			
(1)	d, death rate per year before control	.190	.014	.018
(2)	d′, death rate per year after control..	.063	.007	.009
(3)	p_0, proportion never infected	.20	0	0
(4)	p_f, proportion of fever cases	.11	.07	.04
(5)	p_b, proportion currently infected	.69	.78	.36
(6)	p_i, proportion previously infected	0	.15	.60
(7)	P, parasite rate	.80	.85	.40
(8)	d_0, death rate of never infected.....	.163	.012	.015
(9)	d_f, death rate of fever cases	.253	.019	.024
(10)	d_b, death rate of currently infected...	.187	.014	.018
(11)	d_i, death rate of previously infected .	unknown	.012	.018
	Villages where no treatments were applied (follow-up units 1, 2)			
(12)	d, death rate per year before control	.187	.008	.027
(13)	d′, death rate per year after control..	.124	.004	.017
(14)	F, factor of exogenous decline in d ..	.663	.5	.630

NOTES:

The estimates in table 1 are based on two sources (see references):
A World Health Organization (1975)
B Molineaux and Gramiccia (1980).
Other abbreviations used are:
F figure
T table
p page.

Lines (1) and (2). Source A, T 2, gives the infant mortality rate (IMR) of follow-up units 5 and 7 before and after control. T 10 gives the death rates of age groups 1-4 years, 5-28 years and 29 years and older before and after control. I computed d and d′ for 0-4 years as the weighted mean of the corresponding IMR and death rate for 1-4 years, using the weights 63/240 and 177/240 given in A, T 1. I set d for age groups 5-18 years and 19 years and older equal, respectively, to those given in A, T 10, for age groups 5-28 years and 29 years and older. For the post-control death rates in the two older age groups, I smoothed the data as follows. There were one death in 5-28 years and 12 deaths in 29 years and older, giving a death rate for the two groups combined of 13/(813 + 801) = .008, where 813 and 801 are the sizes of the two groups. I then required $d'(5\text{-}18)/d'(19+) = d(5\text{-}18)/d(19+)$ and $[813 \cdot d'(5\text{-}18) + 801 \cdot d'(19+)]/(813+801) = .008$.

Line (3). $p_0(0\text{-}4) = 1 - P(0\text{-}4)$. $p_0(5\text{-}18) = p_0(19+) = 0$ on the basis of B, p. 121, F 25.

Line (4). B, p. 256, T 28. Fever is defined as body temperature of 37.5°C or more. $p_f(0\text{-}4)$ is the per cent with fever in village cluster 2 at survey 15, age less than 9 years. Village cluster 2 is the only untreated follow-up unit for which data on fever prevalence are presented in B, p. 256, T 28. $p_f(19+)$ is the same for age 9 and older. $p_f(5\text{-}18) = [p_f(0\text{-}4) + p_f(19+)]/2$. These figures on the prevalence of fever differ a little from those quoted above from Molineaux and Gramiccia (1980, p. 258) when "fever cases" are defined in this text because the latter data are pooled over surveys and village clusters.

Line (5). $p_b = P - p_f$ = line (7) − line (4).

Line (6). I set $p_i(0\text{-}4) = 0$. For the two older age groups, $p_i = 1 - P$.

Line (7). Read from B, p. 144, F 38.

Lines (8), (9), (10), (11). Source A, T 6, gives the IMR in the baseline phase according to the infants' parasitological status at the beginning of an interval of observation. Infants who were uninfected had IMR = .202. Infants infected with *P. falciparum* who had fewer than 25 per cent of thick film fields positive for asexual stages of the parasite had IMR = .231. Infected infants who had 25 per cent or more of fields positive had IMR = .311. Now, the greater the proportion of fields positive for *P. falciparum* asexual stages, the greater the probability of fever (B, p. 256, T 29). Hence, I set the ratio .231/.202 = 1.15 equal to the ratio d_b/d_0, and the ratio .311/.202 = 1.55 equal to the ratio d_f/d_0. Then, I assumed, for all age groups,

$$d = p_0 \cdot d_0 + p_f(1.55d_0) + p_b(1.15p_0) + p_i \cdot d_i. \quad \text{(Ta1)}$$

(In principle, d_0, d_f, d_b and d_i could all be computed directly from the Garki data tape.)

For 0-4 years, $p_i = 0$, so (Ta1) determines d_0, since d, p_0, p_f and p_b are given by lines (1), (3), (4) and (5) respectively. From d_0,

$$d_f = 1.55d_0, \quad \text{(Ta2)}$$

$$d_b = 1.15d_0. \quad \text{(Ta3)}$$

For 5-18 years and 19 years and older, (Ta1) is not sufficient to determine either d_0 or d_i. Therefore, I made the further assumption that

$$d_0(19+)/d(19+) = d_0(5\text{-}18)/d(5\text{-}18) = d_0(0\text{-}4)/d(0\text{-}4) = .163/.190 = .858. \quad \text{(Ta4)}$$

In combination, (Ta1) and (Ta4) determine the values shown.

As line (3) shows $p_0(5\text{-}18) = p_0(19+) = 0$, it is important to point out that $d_0(5\text{-}18)$ and $d_0(19+)$ are hypothetical estimates of the death rates of those age classes in the absence of malarial infection.

An unanticipated consequence of the calculation is that $d_i(5\text{-}18) = d_0(5\text{-}18)$. Young people who lose their infection are estimated to have the same mortality as those never infected, that is, their mortality appears to recover to normal once they are freed of infection. However, $d_i(19+) = d_b(19+)$. Older people freed of infection suffer excess mortality that is as adverse as if they were still infected. Qualitatively, these findings seem realistic.

Lines (12), (13). The same procedures and sources were used as for lines (1) and (2), without the smoothing procedure used there.

Line (14). F = line (13) divided by line (12). The ratio .663/.5 of the largest to the smallest F is much smaller than the ratio (.187 − .124)/(.027 − .017) of the largest to the smallest of the corresponding differences between line (12) and line (13); hence, it seems more economical to summarize the exogenous change in mortality as a multiplicative change (multiplication by a factor F) than as an additive change (subtraction of a constant amount).

before control (d). The present section is devoted to the question of how well the model of a multifaceted mcp can predict d′, using only information available prior to control. All the calculations presented here, being based on heroic assumptions and good but limited data, are approximate.

Table 1 gives an internally consistent set of estimated values for the parameters of the model of a multifaceted mcp. The notes to table 1 describe in detail how these values were obtained. In brief, some of the values are taken directly from Garki measurements, and the rest of the values are calculated from measurements under assumptions intended to be reasonable or at least plausible. The measurements are taken from Molineaux and Gramiccia (1980) and from an unpublished report of the World Health Organization (1975), which was used in preparing their study.

A magnetic tape of the Garki project's raw data is available, and careful analysis of those data would make

it possible to replace some calculations or guesses in table 1 with measurements. Because table 1 is intended only as a realistic numerical illustration, I have been content here with existing tabulations.

The most vulnerable assumptions made in computing table 1 are expressed in equations (Ta1) to (Ta4), which are found in the note to lines (8) to (11) of that table. A first assumption is that the ratios d_f/d_0 and d_b/d_0 are the same in all age classes as among infants, and may be estimated from the ratios of death rates among individuals with high and low densities of *P. falciparum* infections. A second assumption is that the ratio d_0/d for the two older populations is the same as the ratio d_0/d for the 0-4 years population. These assumptions are not part of the general model, but are temporary adjuncts that make it possible to obtain numerical estimates of the model's parameters from available tabulations of the Garki data.[7]

When the estimated death rate d_0 of individuals never infected with malaria (line 8) is compared with the post-control death rate d' (line 2) in each population, d' is seen to be lower than d_0. Even if the mcp could restore to all individuals the pre-control mortality of the never infected, it could not lower the mortality of each treated population as far as the observed mortality fell.

At least three factors could possibly explain how the post-control mortality of a population might have fallen below the pre-control mortality of its never-infected subpopulation. First, the multifaceted mcp probably reduced causes of death in the treated villages in addition to malaria. Secondly, exogenous factors probably caused a decline in mortality independently of any mcp. Thirdly, the numbers of recorded deaths might be so low that the difference is due to sampling fluctuation. The evidence pertinent to these factors will be described below.

First, as Vaugelade pointed out (J. Vaugelade, personal communication, 22 January 1987), in the treated villages, the antimalarial drug pyrimethamine was administered in combination with sulphalene, a long-acting sulphonamide (Molineaux and Gramiccia, 1980, pp. 23-25). Independently of malaria control, sulphalene would have reduced mortality, and most strongly among infants and young children. Indeed, comparison of lines (1) and (2) shows that the decline in the death rate, both absolutely and relatively, was largest among children aged 0-4 years. It is impossible to say whether this difference among populations should be attributed to sulphalene or to the mcp.

Secondly, exogenous changes in mortality lowered death rates in villages that were left untreated in both the baseline and intervention phases (lines 12 and 13). Without any intervention, death rates declined in those villages (line 14) to one half to two thirds of the original level. Since the untreated villages received no drugs or other treatments, the effects of malaria control and sulphalene contribute jointly to the difference in mortality between the treated and untreated villages.

Thirdly, the numbers of deaths used to calculate the estimates in table 1 are low (World Health Organization, 1975). For example, if the rates are given as (the number of events)/(the population at risk), the infant mortality rate in follow-up units 5 and 7 was 14/61 during the baseline phase and 9/112 during the intervention phase, while the death rates of those aged 1-4 years during the corresponding phases were 25/142 and 11/194, respectively; the rates $d = 0.190$ and $d' = 0.063$ in lines (1) and (2) of table 1 for children aged 0-4 years are weighted means of these fractions. To test the null hypothesis that there is no difference between the infant mortality rates of the baseline and intervention phases, I assume Poisson sampling of deaths and compute X^2 corrected for continuity, with one degree of freedom, as 6.38, which has a probability between 0.01 and 0.025. For children aged 1-4 years, the difference in death rates is significant beyond the 0.005 level. Thus, there is very little likelihood of no real decline in the death rate of the 0-4 years population associated with the intervention phase. The sample sizes of the other estimates in table 1 are similarly small. While it would be possible to test every asserted difference for statistical significance, it seems more productive, for this example, to suppose that the sample sizes are sufficient for valid inferences. Henceforth, attention will be limited to the first two factors.

The model cannot, and is not intended to, explain either exogenous changes in mortality or effects of a control programme on causes of death unrelated to malaria. At best, it can hope to explain changes in excess mortality associated with malaria. An independent model or measurement is required to estimate other changes in mortality.

Because there is no quantitative information about the effects of sulphalene on mortality in the treated villages, this numerical illustration will consider only the exogenous change in mortality. The exogenous change can be estimated by comparing death rates in the untreated villages before and after control.

Suppose that, in the absence of treatment, the exogenous decline in mortality experienced by the untreated villages would have affected in the same multiplicative way each subpopulation of the treated population. Table 2 gives the hypothetical death rates expected during the intervention phase for each subpopulation, in the absence of multifaceted control, adjusting only for the exogenous factor of mortality decline (lines 15 to 18).

Even after this adjustment, for the population aged 0-4 years, the estimated death rate 0.108 of the never-infected subpopulation (line 15) still exceeds the death rate 0.063 actually observed (line 2) after multifaceted control. This suggests that the sulphalene supplied in combination with pyrimethamine contributed to the decline in mortality of the children aged 0-4 years, or that the exogenous decline of mortality in the treated villages was greater than that in the untreated villages for the population aged 0-4 years, or that the excess of 0.108 over 0.063 could at least partly be due to sampling variability in the estimates of the death rates.

For the populations aged 5-18 years and 19 years and older, the estimated death rate d_0 of the never-infected subpopulation is smaller than the death rate d actually observed after multifaceted control.

The death rates adjusted for exogenous mortality decline (the upper half of table 2) and the proportions of the subpopulations (in table 1) may be combined to give

TABLE 2. PREDICTED MORTALITY FOLLOWING MALARIA CONTROL IN THE GARKI PROJECT

Line	Parameter	Population (age group in years) 0-4	5-18	19+
	Villages where treatments were applied (follow-up units 5, 7)			
(15)	d_0F, adjusted rate of never infected	.108	.006	.009
(16)	d_fF, adjusted rate of fever cases.....	.168	.010	.015
(17)	d_bF, adjusted rate, currently infected ..	.124	.007	.011
(18)	d_iF, adjusted rate, previously infected	unknown	.006	.011
(19)	d′ with multifaceted control success 0.	.126	.007	.011
(20)	d′ with multifaceted control success .5	.112	.006	.011
(21)	d′ with multifaceted control success 1.	.108	.006	.011
(22)	d′ with eradication, compete success ..	.108	.006	.009
(2)	d′, death rate observed after control ...	.063	.007	.009

NOTES:

Lines (15), (16), (17), (18). Subpopulation death rates from lines (8), (9), (10), (11), respectively, multiplied by the factor F of exogenous decline from line (14).

Line (19). $d' = p_0 \cdot d_0F + p_f \cdot d_fF + p_b \cdot d_bF + p_i \cdot d_iF$, based on lines (3) to (7) and (15) to (18).

Line (20). d′ based on text equations (3a) for 0-4 years and (3b) for 5-18 years and 19 years and older, using adjusted death rates from lines (15) to (18) instead of actual rates and $c \cdot s_B = c \cdot s_F = s_b = s_f = 1/2$.

Line (21). d′ based on text equations (3a) for 0-4 years and (3b) for 5-18 years and 19 years and older, using adjusted death rates from lines (15) to (18) instead of actual rates and $c \cdot s_B = c \cdot s_F = s_b = s_f = 1$.

In lines (19) to (21), the subpopulation-specific death rates were first adjusted for exogenous decline by the factor F and then the impact of the mcp was computed using (3a) and (3b). As may easily be verified algebraically and numerically, the results are identical if the impact of the mcp on the original subpopulation-specific death rates is computed first using (3a) and (3b) and the results are then adjusted for exogenous decline by the factor F. The reason is that either way F appears linearly in all terms on the right sides of (3a) and (3b). Hence, our numerical procedure makes no implicit assumption about the order of action of the mcp or the exogenous decline.

Line (22). All individuals have the adjusted death rate of those never infected. Identical to line (15).

Line (2). Estimated actual death rate, from line (2) of table 1.

the predicted post-control death rates (the bottom half of table 2) under various assumptions about the success of multifaceted control or eradication. To facilitate comparison of these predicted rates with those actually observed, table 2 repeats the observed post-control death rates d′.

When the success of multifaceted control is 0 (line 19), the predicted death rate corresponds to no mcp at all, and is the result of applying equation (1) to the estimated subpopulation proportions and the adjusted death rates. For the populations 0-4 years and 19 years and older, no success is predicted to have resulted in death rates higher than those observed. For the population aged 5-18 years, the observed death rate is indistinguishable from that estimated assuming no success.

Setting the success of multifaceted control equal to 1 (line 21) is a hypothetical extreme, because it corresponds to the complete elimination of fever cases and of currently infected cases. This extreme may be viewed as the limit of a highly successful multifaceted control programme; the Garki project, for example, did succeed in reducing the prevalence of infection by 95 per cent or more (Molineaux and Gramiccia, 1980, p. 144). The same set of model parameters may be interpreted to describe an eradication programme just after eradication has been achieved, before the previously infected cases have died out or lost their excess mortality due to prior infection.

For the youngest population, 0-4 years, the greater the success of multifaceted control, the lower the estimated death rate. However, eradication offers no further lowering of mortality beyond that attained by completely successful multifaceted control, because, according to the model, the youngest population has no subpopulation of previously infected.

For the oldest population, 19 years and older, the success of multifaceted control, whether 0 or 1, makes a negligible difference in mortality. The reason for this perhaps surprising finding is that, with no individuals never infected and very few fever cases, this population consists overwhelmingly of individuals currently infected or previously infected. Multifaceted control shifts individuals from the currently infected subpopulation to the previously infected subpopulation. But according to table 1, these two subpopulations are estimated to have nearly identical mortality. Hence, there is no change in mortality with more successful multifaceted control. However, eradication would eventually assure that the entire population was never infected by malaria. This shift would lower the estimated death rate to that actually observed.

Finally, for the population of intermediate age, 5-18 years, increasingly successful multifaceted control and eradication are estimated to cause very small declines in mortality because the adjusted death rates d_0F, d_bF and d_iF of the largest subpopulations are so similar.

The range of estimated death rates for the populations 5-18 years and 19 years and older does include the actual death rate observed after control, but the range estimated for the population aged 0-4 years is too high (as previously mentioned).

In a real use of the model, as pointed out by Rabinovich (J. Rabinovich, personal communication, 26 December 1986), it would be important to carry out a thorough sensitivity analysis to determine how vulnerable the predictions are to sampling or observational errors, such as false positives and false negatives. Since the numbers used here are based partly on bold assumptions, a sensitivity analysis of them is justified only to illustrate the method. Suppose, for example, that the population aged 0-4 years had not 20 per cent never infected (as in line (3)) but 10 per cent, and that 5 per cent had been previously infected (instead of 0 as in line (6)) and 74 per cent were currently infected (instead of 69 per cent as in line (5)). Suppose also that the death rate d_i of the previously infected was 0.012, the same as for the age group 5-18 years in line (11). These assumptions lead to a pre-control death rate $d = 0.183$, slightly below the $d = 0.190$ shown in line (1). The predicted post-control death rates are also slightly lower. The predicted values of d′ for lines (19), (20) and (21) become 0.121, 0.108 and 0.103. Thus, even assuming that half of the infants and young children are false negatives does not lower the predicted death rate with complete success as far as the death rate $d' = 0.063$ observed after control.

The present section began with the question of how much useful information the model could give about the

post-control death rate of a population, given only estimates of the model's parameters prior to control and estimates of the planned control efforts. The numerical example given here suggests that the model can do quite well at predicting the post-control death rate, provided the exogenous change in mortality and the effects of the mcp on other causes of mortality in the treated population are known. The change in mortality that would have occurred in the absence of the mcp can be predicted, prior to control, by an independent theory derived from some other source; or it can be measured, after control, in an untreated population whose death rates are known or believed to behave like those of the treated population. For example, routine vital statistics in an ecologically similar country neighbouring the treated country, or in a yet untreated portion of the treated country, might be used to evaluate exogenous mortality. The effects of the mcp on non-malarial sources of mortality seem more difficult to estimate, especially if those causes are likely to interact with malarial mortality. However, without good information about exogenous and non-malarial programme-related changes in mortality, the model presented here cannot be expected to offer much predictive power.

Conclusion

This section sketches alternative approaches to estimating the impact on mortality of mcps. It then reviews some of the strengths and weaknesses of the models proposed here. Finally, it offers some recommendations for the collection and analysis of data in the intersection of malarial epidemiology and demography. These recommendations can be generalized to efforts to predict the mortality impacts of other major diseases.

Alternative approaches

The essential idea of the models proposed here is that a large part of the heterogeneity among individuals in the effects on mortality of mcps can be accounted for by stratifying the individuals into four subpopulations based on prior or present experience with malaria. For example, a chemotherapeutic programme could be expected to reduce the mortality of a malarial fever case substantially, but the mortality of a never-infected person hardly at all. Stratifying to recognize major elements of heterogeneity could be pushed much further, as has already been done by Manton and Stallard (1984) for human mortality data unrelated to malaria.

In the context of malaria control, as pointed out by Molineaux (L. Molineaux, personal communication, 9 December 1986), one could determine the cause, such as malaria and "other", and other characteristics, such as age, sex and residence, in a sample of deaths. From counts of the population at risk in each cross-classified stratum of age, sex and residence, one could estimate cause-specific death rates by stratum. One could assume that malaria and "other" causes act independently within each stratum, and use the Bernoulli-Makeham procedure to estimate the impact of removing malaria, stratum by stratum. Though independence between malaria and "other" causes of death would be assumed within strata, malaria and "other" causes could be correlated overall (this should be considered as spurious correlation).

This approach demands considerably more, and more detailed, data than the models proposed here. Because of the limited number of deaths observed, the data of the Garki project are not likely candidates for this mode of analysis. The idea deserves to be kept in mind if more extensive sets of data become available.

A second alternative approach, as noted by Dietz (K. Dietz, personal communication, 30 December 1986), would be to begin with an existing dynamical model of malarial transmission (e.g., Bailey, 1982; Dietz, 1986) and consider different death rates for different strata of people. Interventions that shift the numbers of people in different strata would lead to changes in death rates. An advantage of this approach is that it offers the possibility of describing the transient effects of mcps. A possible weakness is that estimates of the parameters of such a model may require more data than the approach taken in this paper.

Critique of the model

Not one but a family of models has been proposed: one model for chemotherapy programmes, one for multifaceted programmes and various possible models for eradication programmes (depending on the function chosen for the decay of excess mortality of the previously infected). Since the underlying ideas of all models in this family are very similar, it is convenient in the following critique to speak of "the model", keeping in mind that various forms are possible.

The model proposed and illustrated here has several strengths: extreme simplicity, a foundation in the biology and epidemiology of malaria and a requirement only for variables that can be measured in the field.

Because the model is extremely simple, it is intellectually transparent. It rests on no hidden simulations, no hidden variables, no complex but obscure equations. The assumptions on which the model is based can be clearly identified. These assumptions can be modified or improved as warranted by additional facts or theories.

A second strength of the model is that it attempts to capture the main biological, epidemiological and demographic features of malaria in the field. The strategy of the model, explicit in equation (1), is to stratify the population into demographically more homogeneous subpopulations based on how the individual is affected by malaria. The structure of the model is guided by what is known of how different kinds of mcps affect the various subpopulations.

Third, the variables in the model have clear operational definitions. Almost all of the variables in the model could be measured directly, or estimated with very few inferences, from the raw data of the Garki project. Knowing in advance what variables are of interest, future mcps could measure directly all the variables, with the exception of an exogenous change in mortality, from data routinely gathered by mcps. Even the exogenous change in mortality could be estimated from routinely gathered vital statistics.

The model also has several weaknesses, some of which are rooted in its strengths. It assumes homogeneity

within subpopulations. It assumes that mcps affect only individuals affected by malaria. It ignores seasonality. With the partial exception of the model for an eradication programme, it compares only a static "before" with a static "after", ignoring the duration and dynamics of the transition. It requires more detailed epidemiological and demographic information than is routinely available now. The model for a multifaceted programme does not predict the sizes of the post-control subpopulations of fever cases and currently infected cases.

The extreme simplicity of the model means that many of its assumptions are only approximations to reality. For example, each of the four subpopulations in equation (1) is treated as homogeneous (possibly after age stratification). In fact, the mortality of a fever case may depend strongly on how often or how long the individual has had fever, but the model omits any duration dependence. The mortality of an infected individual without fever depends on the density of infection, that is, the fraction of blood cells parasitized by malaria, but the model omits any density dependence. The mortality of a previously infected individual without fever or parasites may depend strongly on his or her immune titre, but the model omits any dependence of mortality on titre other than an all-or-none classification as positive or negative. The model omits heterogeneity in residence, as an example of an easily measured demographic variable, although an individual living near a rural swamp is exposed to a different intensity of reinfection from an individual living in a city.

The model treats the mortality of the never-infected population as independent of the mcp, though possibly subject to exogenous changes. However, Gramiccia and Hempel (1972, p. 191) mention that, following a malarial eradication programme in Venezuela, Gabaldon found decreased death rates among infants and young children due to diarrhoea and enteritis, "this being probably due, to some extent, to the action of the anti-mosquito insecticide on flies and other contaminating insects". Similarly, antimalarial spraying of DDT in India led to the near-disappearance of kala-azar in India during the 1950s (e.g., Sanyal, 1982). Thus, an mcp could affect the mortality of individuals never infected by malaria. The model omits any such effect.

The model ignores seasonality in the ecology of malaria. The season in which control measures are applied will materially affect their impact on incidence, prevalence and mortality. As presented, the model considers only averages over a year. However, the model could in principle be adapted to deal with one season as the unit of time, at the expense of additional complexity.

Beyond ignoring seasonality, the model ignores the process and impact of the transition from pre- to post-control. It simply assumes a transition from one steady-state ecology to another. It offers little guidance, for example, about how long to wait before measuring the post-control parameters for comparison with the model's predictions.

It is perhaps both a weakness and a strength of the model that the model requires more detailed epidemiological and demographic measurements than are now routinely provided by most demographic systems of vital registration. The model cannot predict the mortality impact of mcps where the required information is lacking. The model fosters no illusions that the mortality impact of mcps can be predicted without the required measurements or estimates.

However, as Horiuchi pointed out (S. Horiuchi, personal communication, 19 December 1986), when partial information is available, it would be sensible to modify the model to profit from it. For example, if a parasitological survey had been carried out but the prevalence of fever cases could not be estimated separately, it would make sense to modify the model to combine the fever and currently infected subpopulations. Similarly, if a serological survey identified all individuals with recent or current malarial exposure, it would be reasonable to simplify the model further by combining the fever, currently infected and previously infected subpopulations. In short, though the model asks for additional data, it should be adapted for use under real conditions.

The model for a multifaceted control programme does not predict the post-control sizes of the subpopulations of fever and currently infected cases. The numerical example based on the Garki project circumvented the need to predict the sizes of these subpopulations by considering a range of possible effects of the mcp (lines (19)-(21) of table 2). For some practical purposes, such a procedure may be sufficient. However, it would be useful to be able to predict the fever and currently infected subpopulations that will result from a multifaceted programme on the basis of mathematical models of malarial transmission (e.g., Gonzalez-Guzman, 1980; Bailey, 1982; Dietz, 1986). The further development of such dynamic models, suggested above as a second alternative approach, is really a necessary complement to the simple models proposed here.

Recommendations

Several recommendations for action follow from this study.

First, it would be informative to analyse the raw Garki data, and existing data from any other studies with the required detail, to see whether the predictions of the model improve or deteriorate when the variables in the model are measured more directly, with a smaller component of conjecture than here.

Secondly, if the results of these analyses are encouraging, it would be informative to test the model further in countries where malaria may be a major cause of death. One would need to record, for each dying person, whether the person was at the time of death feverish, or was known (preferably on the basis of microscopy) to be malarially infected, or had been previously malarially infected. If it is impossible to collect such information on a national scale, it might be possible to collect it in suitably designed samples of deaths. The data collection could be patterned after recent efforts to assess the causes of deaths in Bangladesh (Zimicki, 1986) and Senegal (Garenne and Fontaine, 1986); these efforts rely entirely on non-medical personnel to collect data from

the field. Sample surveys could be used to estimate the prevalence of malarial fever, malarial infection and prior malarial infection. These prevalences, in combination with data on the cause of death, give estimates of the specific death rates and sizes of the subpopulations described by the model. Countries considering mcps could then use the model to try to anticipate the impact of malarial control on death rates. The considerable effort and expense required to use the model would be far less than that required for the Garki project and could largely be absorbed in the planning phase of an anticipated mcp.

Thirdly, if the cumulative experience with the model remains encouraging, the model's simple strategy, namely, stratifying the population into epidemiologically more homogeneous subpopulations, based on the biology of the disease, might be extended to other diseases with a major impact on mortality.

This analysis will remain an idle exercise unless people in the countries affected by malaria use it, improve it and adapt it to their own needs. Able epidemiologists in the countries where malaria is endemic appear to be in short supply. The steering committee of the scientific working group on epidemiology of the Special Programme for Research and Training in Tropical Diseases (sponsored by the United Nations Development Programme, the World Bank and the World Health Organization) described the special problems of epidemiology in the endemic countries (World Health Organization, 1983, pp. 3 and 4) as follows: ". . . of all disciplines needed in public health in the tropical countries, epidemiology is perhaps the most severely neglected. If the tools that are being developed . . . are to be put to use, then the epidemiological [and, I would add, demographic] capabilities of the disease control programmes in endemic countries must be strengthened. . . . a vital function of the epidemiologist in developing countries is often that of obtaining basic data." The future of the ideas in this paper lies with people in the endemic countries who can put the ideas to work and make them evolve.

Notes

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[2] However, measles and other causes of death may not always be complementary as McGregor (1964) reported for measles and malaria; see, for example, Pison and Langaney (1985).

[3] It might be argued that the spraying of households with DDT and other toxic residual insecticides, and the mass administration of potent antimalarial drugs, some with adverse side-effects, open the possibility that a successful mcp might actually raise death rates eventually. The potential benefits so far outweigh the minuscule potential risks of mcps that this theoretical possibility will not receive further analysis here.

[4] When it is possible to distinguish malarial fevers from fevers of other origins, then only malarial fevers should be counted. When this distinction cannot be made, then the clinical definition quoted above from Breman and Campbell (1986) should be followed.

[5] These definitions refine the proposal of Breman and Campbell (1986) that "a patient with probable [fever case] or confirmed [parasitemic case] malaria who dies becomes a death due to malaria". The refinement seems justified by the likelihood that the death rate among fever cases is higher than the death rate among parasitemic cases without fever, and by the probability that these two groups of individuals will be differently affected by different tactical variants of mcps.

[6] An interesting and important question, which I shall not attempt to answer here, is what happens to mortality if a hitherto successful mcp is interrupted. This model is premised on the assumption that the mcp is sustained.

[7] It is interesting to compare the fraction of deaths among children 0-4 years from or with malaria in rural northern Nigeria, according to the figures in table 1, with an estimate of the fraction of infant and child deaths in which acute malaria can be incriminated as the cause of death [Bruce-Chwatt, 1952, p. 198] in Lagos, Nigeria. From lines (1) and (8) of table 1, the fraction of deaths from or with malaria prior to the Garki mcp is $1 - d_0/d = 1 - 0.163/0.190 = 0.14$. Analysing records of autopsies performed on children in Lagos during the years 1933-1950, Bruce-Chwatt (1952, pp. 198-199) identified acute malaria as the cause of death in 9 per cent of infants and 14 per cent of children aged 1-4 years. Considering the differences in origin between the Garki project's population-based data and Bruce-Chwatt's autopsy records, the agreement is remarkable, though possibly coincidental.

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ANNEX

Symbols

Symbols without a prime refer to pre-control conditions. Symbols with a prime ($'$) refer to post-control conditions.

c	competing risk control coefficient
D	coefficient that describes the decay of excess mortality
d	death rate of the population
d_0	death rate per year of individuals never infected with malaria
d_b	death rate per year of individuals currently infected, without fever
d_f	death rate per year of fever cases
d_i	death rate per year of previously infected individuals
F	factor of exogenous decline in mortality
f(t, n)	decay function for excess mortality of previously infected persons
P	parasite rate = $p_f + p_b$
p_0	proportion of individuals never infected with malaria
p_b	proportion of individuals currently infected, without fever
p_f	proportion of fever cases
p_i	proportion of previously infected individuals
S	spleen rate or sero-positive rate or serum rate = $P + p_i$
s_b	success in reducing the excess mortality of parasitemic cases
s_B	success in reducing the proportion of parasitemic cases
s_f	success in reducing the excess mortality of fever cases
s_F	success in reducing the proportion of fever cases
s_P	success in reducing the parasite rate
t	years after eradication

INTERRELATIONSHIPS BETWEEN CHILD SURVIVAL AND FERTILITY

United Nations Secretariat *

SUMMARY

Theoretical considerations, largely supported by empirical research findings, confirm the interdependence of child survival and fertility. Levels and trends in child survival are negatively associated with levels and trends in fertility. However, the prevalent direction of causation, its mechanisms, timing and strength, are not identical in different populations. Patterns of demographic transition show that improvements in child survival typically precede sustained fertility decline. Thus, interventions to improve the health of children will ultimately be followed by a decline in the birth rate. The actual fertility consequences of such interventions will depend not only on the type of intervention but also on the prevalent family-building strategy and the nature and scope of family-planning programmes in a given location. In particular, the ready availability of family-planning services is seen to intensify any fertility decline resulting from improvements in child survival in most settings by providing parents with greater control over their fertility.

INTRODUCTION

Do reductions in infant and child mortality moderate fertility? A correct answer to this question is needed not only for its theoretical importance in the conceptualization of the demographic transition, but also for very practical reasons. Thus, knowledge of the linkages between mortality decline, use of family planning and changes in fertility offers the policy maker the opportunity to achieve greater gains in the health and economic well-being of the population within a given budget through the implementation of programmes designed to enhance or neutralize these linkages.

However, while current global strategies in the health and family-planning fields are based on the assumption that improved child survival is a major prerequisite of sustained fertility decline,[1] empirical research findings suggest that fertility responses to reductions in infant and child mortality vary widely in timing and strength. This lack of consistency in empirical results clearly impedes the design of integrated health and family-planning policies. There may be two major interrelated reasons for the variability of empirical results. First, even the prevalent direction of causation, its mechanisms, timing and strength, may differ between populations. Secondly, in this particular research domain empirical results are likely to be especially sensitive to the choice of variables and statistical techniques.

The present article[2] explores a variety of relationships between child survival and fertility behaviour within environments that differ according to the socio-cultural correlates of demand for children, the prevailing mortality pattern and the availability of family planning. A range of probable effects of improved child survival on fertility for different settings are derived from logical argument and empirical evidence drawn from adequately designed studies. This permits the formulation of a few policy guidelines and the identification of promising areas for future research.

CHILD SURVIVAL: FERTILITY RELATIONSHIPS IN THE SOCIETAL CONTEXT

Most of the research on the effects of infant or child mortality on fertility addresses the question of how parents respond to the experience or the expectation of a child death. The child survival hypothesis lies at the core of both theoretical and empirical studies in this domain. In its simplest form, the hypothesis states that experience with or fear of child mortality may lead parents to have additional births either to "replace" those who have actually died or as "insurance" against expected deaths. The implication then follows that improved child survival will increase the motivation to limit births and subsequently lead to fertility decline (Taylor, Newman and Kelly, 1976). Thus, the child survival hypothesis, which emphasizes conscious fertility responses on behalf of parents to mortality conditions they face, deals with volitional (reflexive) effects which are likely to operate at the familial level through the supply of children when family size goals remain stable and fertility is under a couple's control.

However, this hypothesis ignores several potentially important aspects of the relationship between child survival and fertility. In fact, some of the ways child survival affects fertility transcend the individual couple in

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that they relate to the emergence and acceptability of qualitatively different types of fertility behaviour rather than to quantitative effects appearing at the level of individual families. In addition, at the familial level, improvements in child survival may affect not only the supply of surviving children but family size preferences as well. Finally, the mortality-fertility relationship goes in both directions. For instance, when appropriate methods of fertility control do not exist or are not known, too frequent and too many births may substantially increase rates of infant and child mortality via both physiological and volitional mechanisms.

The proposed analytical framework is based on the concept of "family building", which is conceived here as the process by which an individual family is formed up to some critical point in time through the sequence and timing of births and deaths. Because improvements in child survival have such different implications for family building in contracepting and non-contracepting societies, a basic distinction is drawn here between a process of family building which is dictated by forces outside parental control—"family building by fate"—and one which is subject to parental controls—"family building by design". Societies in which the family-building process is the outcome of fate for the individual family may none the less have developed collective strategies to assure child survival, such as prolonged breast-feeding and post-partum sexual abstinence, which parents conform to but which they do not wilfully manipulate. In many settings, the two processes, family building by fate and family building by design, co-exist either in different population subgroups and/or even within the same family at different points in its life cycle. Parental intervention in the family-building process can occur not only through the practice of family planning but also through the quality of care parents provide their children. While some might call such parental action "family building by design", it is discussed here as an aspect of "family building by fate" because, from the point of view of the individual family, any child death resulting from such inadequate care is likely to appear as a cruel blow of fate rather than an event over which they had any control.

Improvements in child survival generate five distinct but closely interdependent types of changes in patterns of reproduction. Four of them result in lower levels of fertility, while the fifth operates in the opposite direction. These five effects are depicted in figure I in the context of the two main types of family-building strategies just described—"family building by fate" and "family building by design".

"Family building by fate", which is synonymous with natural fertility or the absence of parity-specific fertility control, characterizes all populations prior to the beginning of the demographic transition. The first and potentially most important effect of improvements in child survival on fertility—the "transition effect"—is to increase the predictability of the family-building process to the point that parents can perceive the possibility of influencing future events themselves, thus making the concept of family planning meaningful. This first effect is shown in figure I as the effect which stimulates the process of change from "fate" as the family-building strategy to "design".

A second effect on fertility of improvements in child survival—"the physiological effect"—is to lengthen inter-birth intervals in breast-feeding populations. This effect will be strongest in natural fertility populations and will become weaker in the course of the transition from natural to controlled fertility.

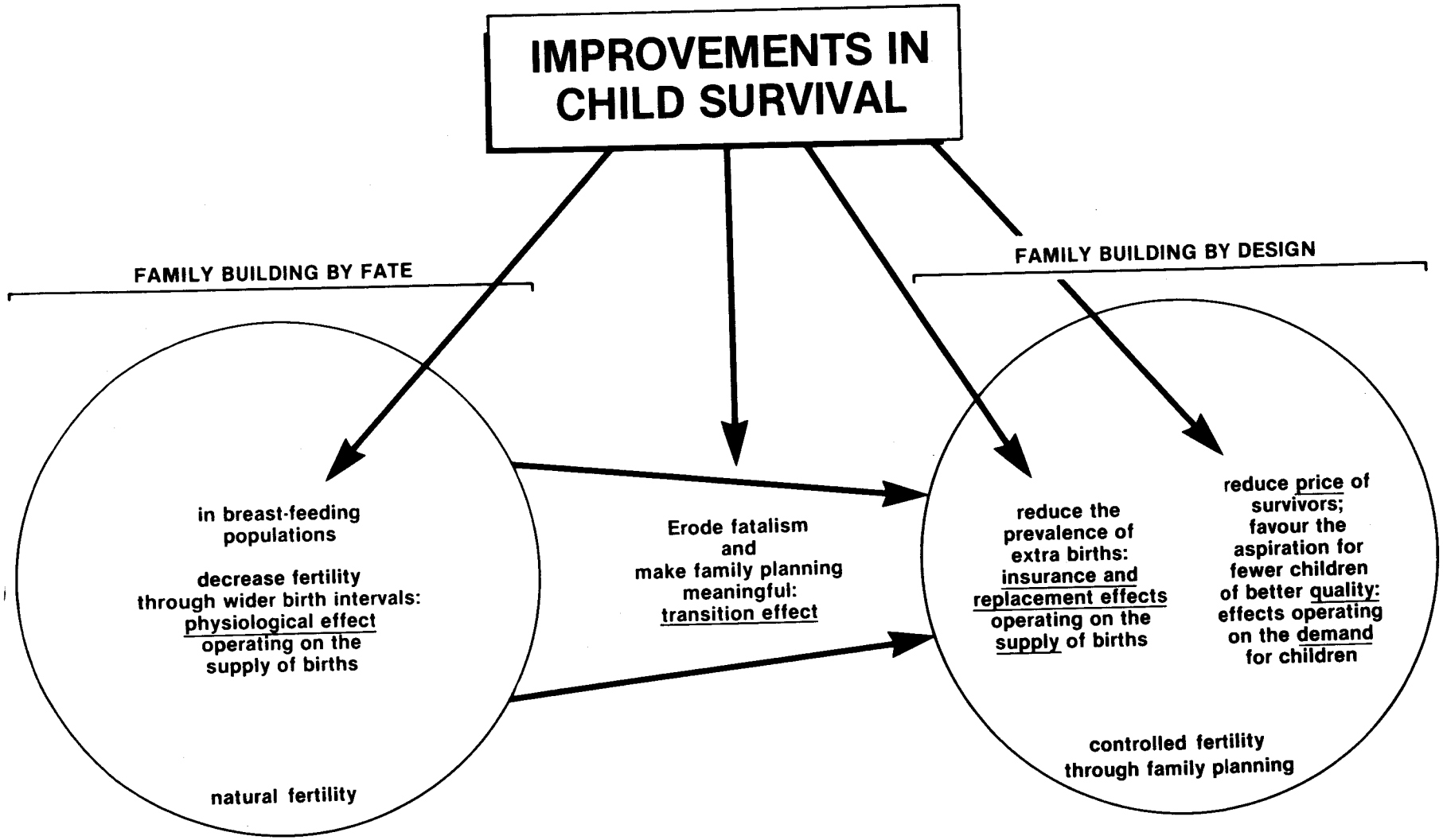
The third, fourth and fifth effects of improvements in child survival on fertility operate within settings where family planning is practised—one effect operates on the supply of births and two on the demand for surviving children. When parents are able and willing to adjust their childbearing plans to actually experienced or anticipated child losses, improvements in child survival will cause decreases in fertility even if family size goals remain unaltered. This is called the "supply effect" and can be achieved through "insurance behaviour" when child survival is still risky and reversible family-planning methods are not widely available or through "replacement behaviour" when child survival is relatively certain after some fairly young age and where reversible family-planning methods are widely available. The "quality effect" of improvements in child survival involves a reduction of the number of surviving children desired as future returns from investments in the education and health of fewer ("higher quality") children become more secure. Finally, the "price effect" involves the reduction in average cost of a surviving child as child survival improves, which, under certain circumstances, may induce parents to revise upwards their family size goals.

Strategies adopted by parents in building their families are conditioned by a vast array of interlinking factors which can be broadly subdivided into two groups: (*a*) societal (structural) conditions and (*b*) parental characteristics. Many factors which have been identified as major determinants of family-building strategies are seen to operate on both the societal and family level, and thus can be examined empirically at either the aggregate or individual level depending on the focus of analysis and the type of data available. Indeed, for many of these factors, the societal level effects are nothing more than a simple aggregation of individual level effects. However, child survival, which is the central factor of interest in the present study, is not such a factor, thus making the analysis of its effects more difficult.

First, the family strategy may be influenced not only by the family's past experience with infant and child deaths but also by parental perception of children's survival chances in the future. Secondly, the timing of changes in fertility behaviour induced by specific improvements in mortality conditions, as well as the strength of the response, are not identical in different populations. The most important intervening factors are the socio-economic setting which influence demand for surviving children and family-planning availability.

The concept of demand, as used here, encompasses the family size goal, expressed in terms of the desired number of surviving children, and "the relevant age", defined as the age up to which a child's survival matters to his/her parents. Under particular mortality and

Figure I.


IMPROVEMENTS IN CHILD SURVIVAL
FAMILY BUILDING BY FATE
FAMILY BUILDING BY DESIGN
in breast-feeding populations
decrease fertility through wider birth intervals: physiological effect operating on the supply of births
natural fertility
Erode fatalism and make family planning meaningful: transition effect
reduce the prevalence of extra births: insurance and replacement effects operating on the supply of births
reduce price of survivors; favour the aspiration for fewer children of better quality: effects operating on the demand for children
controlled fertility through family planning

family-planning conditions prevalent family-building strategies will be dictated by these two characteristics of demand. Thus, the higher the family size goals, and the higher the "relevant age", the more families will be inclined to insure against expected child deaths through increased births (sometimes pushing their fertility close to the biological maximum) rather than waiting to replace those babies who actually die. While other dimensions of demand may also be pertinent, such as the strength of demand, preferences concerning the gender composition of the family and timing and spacing of births, only occasional references to them will be made in this paper.

The formulation by parents of a family size goal emerges from a balancing of costs and values of children over time (preferences interacting with resource constraints). Costs of children include direct costs of child rearing (such as food, housing, clothing, schooling); the indirect (opportunity) costs of parental time and expenditures related to a child's transition to adulthood (such as the allocation of land, housing and dowries). The values of children to parents include the economic benefits they provide such as labour services, old-age support and risk insurance, as well as various non-economic benefits such as social status, political power, physical security, continuation of the family line and emotional fulfilment. The distribution of the values over time will determine the "relevant age". For example, in settings where children are particularly valued by parents for old-age protection, the relevant age would have to be at least 20 and probably higher, whereas in settings where child labour is important but social security is available from other sources, the relevant age might be lower.

Hence, the societal correlates of demand for surviving children include economic, institutional and socio-cultural factors. Among the economic factors should be included income level, mode of production and land and income distribution.[3] Among the socio-cultural factors, the extent of urbanization, the family and kinship structure,[4] the system of beliefs[5] and the role of women[6] in society would be particularly important. Among the institutional factors which should be included are the system of resource ownership and intergenerational transfers and the availability and the distribution of public resources, including education and family-planning services.

The abundant body of literature in this domain, reviewed elsewhere (see United Nations, 1987a), provides the information for a rough classification of broad geographic regions into settings with similar economic, institutional and socio-cultural characteristics and, thus, similar patterns of demand for surviving children.

Table 1 presents a breakdown of 10 different settings within the developing world—five distinct regions which share certain socio-cultural characteristics are presented separately for rural and urban areas. The regions are (*a*) Latin America, (*b*) East Asia, (*c*) Southeastern Asia, (*d*) West and South Asia and North Africa and (*e*) sub-Saharan Africa. For Europe, two types of pre-industrial settings—one for urban Northern Europe and one for rural Eastern and Southern Europe—are contrasted with the modern developed countries as a group. These 13 settings provide a full range of scenarios which can be later compared when the findings from the empirical studies on the mortality-fertility relationship are presented.

TABLE 1. CLASSIFICATION OF PLAUSIBLE COMPONENTS OF DEMAND FOR SURVIVING CHILDREN BY REGION AND/OR SETTING

Region/setting	*Family size goals*	*Relevant age*
Pre-industrial N-W Europe—urban	L to M	M
Pre-industrial E-S Europe—rural	M to H	H
Modern LDCs		
Latin America[a]		
rural	M	H
urban	M	H
East Asia		
rural	M	E
urban	L	E
Southeastern Asia		
rural	M	H
urban	M	H
W. Asia, S. Asia, N. Africa		
rural	H	H
urban	M	H
sub-Saharan Africa		
rural	E	M
urban	H	H
Modern MDCs[b]	L	n.a.

Sources: United Nations (1987a) and (1987b).

NOTES:

Low (L)	1-3	10 years
Medium (M)	4-5	About 15
High (H)	6-7	About 20
Extremely high (E)	Over 7	30 or higher

[a] Except Temperate South America, which is considered similar to the modern developed countries.
[b] Including Temperate South America.

For most settings, rough family size goals can be clearly drawn from expressed family desires recorded in recent fertility surveys (see United Nations, 1987b). Since empirical research directly addressing the issue of the "relevant age" is lacking, its values are tentatively assessed proceeding from the simplistic assumption that the "relevant age" can be determined by the specific type of reward from children which is known to be the most important in a particular setting. In fact, as can be seen from table 1, in all developing settings except East Asia and rural Africa, the relevant age is assumed to be about 20. In East Asia, where the preference for a surviving son is strong and where particular importance is given to children outliving their parents, the relevant age is likely to be 30 years or more. In parts of rural Africa, however, relevant age would be lower because child labour may be of particular value to their parents while old-age support is less crucial because of the availability of support from the larger kin group. This was also the case, for different reasons, in pre-industrial Northern and Western Europe, where poor relief and retirement contracts provided a form of insurance to parents in old age, but where child labour was important to parents in both

urban and rural settings. The pre-industrial rural setting of Eastern and Southern Europe is most comparable to many rural settings in today's developing countries. The "relevant age" is not a useful concept in the developed countries today where children's value does not vary over time as much and is primarily non-economic.

In settings where parents, on average, desire fewer births than would be implied by natural fertility, the availability of family-planning services is an important factor determining not only actual fertility at any point in time but also the extent and speed of fertility decline as child survival improves. Models that simulate the potential effects of child survival improvements on fertility usually assume perfect fertility control. In an imperfect world, however, the actual decline in births will be less than that predicted in the models and will depend on the availability, reversibility and efficiency of family planning. The greater the choice of methods, in particular reversible and efficient methods, and the lower their costs, the larger will be the decrease in birth rates in response to any given improvement in child mortality.

In the past 20 years, there have been many empirical investigations of the impact of mortality on fertility and even several exhaustive reviews of the literature (Schultz, 1976; Mauldin and Berelson, 1978; Preston, 1978; Heer, 1983; Schmitz, 1985). Over time, the quality of data has improved as has the sophistication of researchers in application of statistical methodologies. None the less, no studies to date have used comparable investigative techniques in different settings in such a way as to permit comparisons of effects between settings. Schultz (1976) pointed out this gap 10 years ago but, unfortunately, in the ensuing 10 years no studies have been undertaken to address this point, despite the fact that the general topic of mortality-fertility linkages continues to occupy an important place in the empirical literature.

Empirical studies of this relationship fall into two broad categories: (*a*) aggregate studies that are based on samples consisting of national or subnational averages, usually using census or registration data, and (*b*) individual level studies that are based on sample survey observations drawn from the reproductive experience of individual women. Studies based on aggregate data have one important advantage over individual level studies: the potential for measuring the full impact of mortality on fertility and thus for addressing the question about the overall implications of improvements in child survival for fertility and population growth. Because many of the hypothesized effects of changes in mortality on fertility work through changes in environmental conditions rather than through individual experience, studies based on individual data alone, which do not incorporate community-level effects, can only hope to measure accurately the "physiological effect" and the "replacement effect".

However, extreme caution must be used in interpreting aggregate level results because they cannot measure the response of birth rates to changes in mortality unless a time dimension is included in the analysis (Schultz, 1973). Instead, these data tend to measure the effect on fertility of relatively long-term and stable inter-country or interregional differences in environment, geography and development. These results generally show that the measured relationship between mortality and fertility is statistically significant and of the appropriate (positive) sign, but the size of the measured effects and their implications for population growth vary enormously.

While precise estimates of aggregate fertility effects cannot be derived from existing studies, some of these studies provide suggestive evidence of the ways in which certain characteristics of the setting, such as development level (Friedlander and Silver, 1967; Janowitz, 1971; Anker, 1978), region (Anker, 1978; Cutright, 1983) and family-planning programme strength (Mauldin and Berelson, 1978), can affect the mortality-fertility relationship (for details, see United Nations, 1987a). In particular, there is convincing evidence that the strength of the relationship is enhanced in settings with strong family-planning programmes. Therefore, family planning is seen to play an important role in reducing the stimulating effects of mortality decline on population growth through a quickening of parents' fertility adjustment to their changing mortality environment.

However, aggregative studies are neither capable of discriminating among partial effects nor of providing unbiased estimates of the full impact of child survival on fertility.[7] Hence, the strength of the "transition", "insurance" and "demand" effects remains unmeasured, and perhaps even unmeasurable. Simulation of these effects may, to some degree, substitute for the lack of direct empirical evidence. A simulation will be used in the discussion of the "transition effect" and in order to illustrate the plausible range of "insurance effects" in different socio-economic and mortality conditions. The simulation of the impact of improvements in child survival on the demand for children exceeds the bounds of the present paper because it requires the simultaneous specification of subtle "price" and "quality" effects, while only very rough hypotheses about the operation of each of these two effects can be derived from the available literature.

However, despite their more limited potential scope, individual studies do have their own advantages, including often the superior quality of the data, the availability of data for necessary statistical controls and the greater environmental homogeneity of the population from which the sample is drawn. This is why they will be widely used in the discussion of supply effects.

The review of specific (partial) mortality-fertility interactions is presented below in the order that corresponds to the course of the demographic transition, beginning with the physiological effect—a mechanism likely to be most prominent at the earliest phase of the transition.

Physiological effect

Even in the absence of birth control, mortality levels can have a pronounced influence on fertility levels. In populations where breast-feeding is common and extends for more than a few months, its ovulatory-suppressant effect results in longer average intervals to the next birth if the previous child survives the breast-feeding period than if it dies within that period. In other words, follow-

ing the death of an infant, breast-feeding is discontinued and ovulation is likely to resume sooner than would otherwise have been the case, resulting in an increased risk of pregnancy early in the post-partum period if contraception is not practised. This mortality-induced shortening of inter-birth intervals is hereafter called the "physiological effect". An increase in infant survival would tend to widen inter-birth intervals through this physiological effect and result in a decline in period fertility rates.

The physiological effect is enhanced in cultures that observe post-partum sexual abstinence, which exceeds the duration of lactation-induced amenorrhoea. In many parts of sub-Saharan Africa, where prolonged breast-feeding is prescribed and sexual intercourse during the lactational period is forbidden, the physiological effect is culturally built into behaviour patterns (Ware, 1977). Here, the potential impact of an infant's death on inter-birth intervals is at its maximum. There is definite evidence that, in these settings, post-partum abstinence is practised specifically to ensure that the supply and quality of breast milk are adequate (Caldwell and Caldwell, 1977, 1981) and thus to enhance a child's survival chances.

In every developing country, the potential physiological effect is likely to be much smaller in urban areas than in rural areas because breast-feeding durations have been found to be significantly shorter for urban than rural populations and still shorter for inhabitants of metropolitan areas (Ferry and Smith, 1983). Even some urban contact may be sufficient to reduce breast-feeding (Knodel and Debavalya, 1980). Declines in the prevalence and duration of breast-feeding have been observed in East and South East Asia, as well as in Latin American countries where breast-feeding used to be widespread and prolonged (Bulatao, 1984). In these settings, the physiological effect is likely to be much weaker among younger cohorts of women. In fact, in the case of Latin America, the high level and speed of urbanization has left little room for any pronounced physiological effect of increased child survival on fertility. In some countries of the region, the average length of breast-feeding does not significantly exceed the duration of the post-partum amenorrhoea which would occur in the absence of lactation (World Fertility Survey, 1984).

The quantitative impact of variations in infant mortality rates on total fertility rates through these interval effects can be estimated with a model developed by Perrin and Shepps and modified by Preston (1978). The model shows that, despite the major influence of child mortality on fertility in breast-feeding natural-fertility populations, it is impossible for fertility to make a fully compensating response to variations in mortality through this non-volitional mechanism for the simple reason that the interval to the next birth cannot be reduced to zero owing to the occurrence of a child death. The fall in fertility resulting from an increase in child survival will be greater the longer the breast-feeding period and the wider the practice of post-partum abstinence (as in West Africa), but in no case will this type of fertility adjustment fully compensate for the decline in mortality which caused it. The maximum value of this compensatory response owing to the physiological effect would be 50 per cent if the average inter-birth interval were about 2.5 years. Thus, up to 50 per cent of the increase in the net reproduction rate caused by an increase in child survival could be eliminated through compensating reductions in fertility, although a compensation of 25 to 30 per cent would be more typical (Preston, 1975, 1978). In other words, over the course of a woman's reproductive life, 1,000 fewer child deaths would be associated with about 250 to 300 fewer births in populations where breast-feeding is prolonged.

In all this discussion, it should not be forgotten that the relationship between fertility and mortality goes in both directions. In natural fertility populations, the impact of high fertility on infant and child mortality may be much more significant than the effect of infant mortality on fertility as implied by the physiological effect just discussed. Results of a number of studies carried out in developing countries support the conclusion that too closely spaced and high parity children are at significantly higher risks of death than children born after longer intervals or children of low parity births (Acsádi and Johnson-Acsádi, 1986; Hobcraft, McDonald and Rutstein, 1985). Thus, in certain circumstances, the volitional spacing and limitation of fertility may be the pre-condition for improvements in child survival rather than the other way around.

Inadequate care

The preceding presentation is based on the assumption that the loss of a child is always undesirable from a family's point of view, and, consequently, that infant and child mortality are due purely to factors beyond an individual's control. Indeed, a scarcity of food and other essential resources and a lack of motivation and/or use of family planning could result in the "inadequate care" of "unwanted" children or some other specific parental behaviour affecting infant and child mortality may contribute to this relationship. In such circumstances active societal interventions to improve children's survival chances, in the absence of other necessary social and economic improvements, may be unconsciously sabotaged by families through inadequate care. While exogenous environmental influences such as famines, safety of water and the nature of endemic diseases determine a background level of mortality, the society as a whole and, more particularly, parents have great scope for modifying the chances of children's survival. Polgar (1972) pointed out that exogenous changes in mortality alone cannot explain the extreme closeness of birth and death rates for all of human history prior to the onset of the demographic transition. This leads to the implication that pre-transitional societies must have developed collective strategies to assure slower rates of population growth. Most of these strategies would have to have been directed at child survival (Polgar, 1972; Vishnevsky, 1976; Pavlik, 1979).

Child rearing always requires the allocation of scarce resources, including the allocation of time and emotional care. Pre-transitional societies differ from each other both in terms of the sources of these resources (parents

versus extended kin or even larger groups of people) as well as in terms of the availability of alternative opportunities for resource allocation. Thus, if parents themselves bear most of the costs of child rearing and have access to opportunities to maintain or improve their own well-being, resource constraints will make them reluctant to rear too many children. The ways in which parents can affect infant and child mortality have been documented in various studies (Shorter, 1977; Ware, 1977; Scrimshaw, 1978, 1983). Evidence of the practice of infanticide, abandonment, neglect and the selective provision of food and medical care to children have been drawn from historical and contemporary data from all major geographic regions. All these practices result in variations in child survival which are and could be consolidated into two broad categories: (*a*) infanticide or overt killing and (*b*) "inadequate care". "Inadequate care" would include child abandonment as well as less evident forms of neglect in the areas of feeding, sanitation, medical care, supervision and attention. Scrimshaw (1983) defines such inadequate care in relative terms: "neglect . . . occurs only when an infant or child receives *less* than the family might be able to provide, and less than the family members know should be provided".

The most persuasive evidence for the practice of "inadequate" or "differential" care emerges from studies of sex differentials in mortality and morbidity in societies where strong sex preferences are known to exist (Bhatia, 1983; Wyon and Gordon, 1971; Kimmance, 1972; Welch, 1974; D'Souza and Chen, 1980; Ware, 1981; Jensen, 1982; Scrimshaw, 1983).[8] In addition to children whose gender is less desired, children of high birth order and children with short intervals between them may be vulnerable to differential care. While there is a scarcity of appropriate studies, it is highly likely that differential care accounts for at least part of the well established positive association between the probability of child death and birth order in high fertility populations (Scrimshaw, 1978, 1983; Kunstadler, 1978).

Hobcraft (1987) found that across 34 developing countries a birth within 12 months after the index birth raises the overall average risk of dying between ages one and five by at least 77 per cent. While the first child in a closely spaced pair may be more vulnerable because of the premature termination of breast-feeding, the second in the pair may be more vulnerable to other forms of differential care. Thus, in the context of natural uncontrolled fertility, the need to adjust family size, its composition and the timing of births to concrete circumstances may result in inadequate care of some children. However, if such behaviour were not culturally tolerated, it could not represent anything more than occasional deviations from the "normal pattern". Examples of cultural norms that would make inadequate care of unwanted children morally acceptable would include norms relating to child feeding and health care, a view of children as "non-persons" and a perception of the benign nature of death in infancy (the perception of a dead infant/young child as an "angelito")—norms that were built into the texture of societies as distant as medieval Western Europe (Aries 1962; Shorter, 1977; Badinter, 1980), Latin America (Scrimshaw, 1983) and sub-Saharan Africa (Quesnel, 1984).

Transition effect

While inadequate care might play a substantial role in adjusting the number of children in the family to its resource constraints under the régime of natural fertility, precarious environmental conditions were responsible for most health hazards. The attitude that events in general are outside an individual's control is widely recognized as an important characteristic of pre-industrial societies. The high incidence of fatal diseases in such societies surely played a role in shaping fatalism in many spheres of life, including fertility behaviour. High and fluctuating mortality makes it practically impossible for any couple to predict with confidence how many children will survive from a given number of births.

A demographic perspective can demonstrate how changes in mortality create the environment necessary for the fertility transition. Improvements in infant and child survival increase the predictability of achieving a particular family size from a given number of births. Table 2, which is based on McNicoll's (1986) simulation exercise, illustrates how a twofold increase in life expectancy calls forth profound changes in average distributions of survivors out of the same number of births. The distributions of families with six births according to their number of surviving children by age 20 are compared between a high mortality régime (life expectancy at 25 years) and a moderate mortality régime (life expectancy at 50 years). Under the high mortality régime, the number of surviving children from six births is highly unpredictable. Almost three quarters (73 per cent) of all six-parity women are likely to lose half or more of their children in infancy or childhood and, thus, a family size goal greater than three surviving children would be unattainable for most families. A considerable proportion (16 per cent) of six-parity women would find themselves either childless or with only one surviving child. In these circumstances, a family would require significantly more than six births to have a reasonable probability of achieving four or more surviving children. However, pre-transitional mortality and morbidity conditions would prevent many women from bearing more than six children. Such conditions are clearly not conducive to the fixing of numerical family size goals.

When survivorship rates rise enough for life expectancy to double from its pre-transitional level, the situation changes drastically (see table 2). Under these conditions, only one seventh (14 per cent) of all six-parity women would lose half or more of their children, none of them would be likely to have less than two surviving children, while most (86 per cent) would see from four to six of their children attain adulthood. Moreover, under this régime of moderate mortality, a considerable proportion (21 per cent) of six-parity women would experience no child deaths, while almost no six-parity women would be immune from this experience in the high mortality régime. A family size goal fixed in terms of "no less than" four surviving children becomes attainable for the

TABLE 2. AVERAGE DISTRIBUTION OF FEMALES WITH SIX BIRTHS BY NUMBER OF CHILDREN SURVIVING TO AGE 20 UNDER HIGH AND MODERATE MORTALITY RÉGIMES[a]

Number of children surviving to age 20	*High mortality régime (e_0 = 25) (percentage)*	*Moderate mortality régime (e_0 = 50) (percentage)*
0	3	0
1	13	0
2	27	3
3	30	11
4	19	28
5	7	37
6	1	21
TOTAL	100	100

Source: McNicoll (1986). Based on "West" family of model life table.

[a] Assuming that the probability of death is independent of the birth order.

overwhelming majority (86 per cent) of parity-six couples. Thus, improvements in child survival create a demographic environment in which the relationship between total births and total surviving children becomes predictable enough, for the individual family, to permit the conscious setting of family size goals. Support for this hypothesis can be found in the World Fertility Survey data, which show that it was in many of the high mortality countries that significant proportions of women were unable to respond to the question about desired family size (United Nations, 1987b).

The overall probability of survival to a particular age is not the only feature of the mortality régime which conditions the emergence of conscious family planning. First, the relationship between childbearing and family size will be more predictable and family planning will be more feasible the more the risks of death (for any given survival probability) are concentrated in a narrow time interval shortly after birth. Alternatively, the longer the child is exposed to significant risks of death, the less predictable is the relationship between childbearing and family size and the less effective and less attractive will be family planning. Secondly, family planning will be more attractive the greater control parents themselves exercise over their children's fate. At the aggregate level this feature of mortality conditions becomes apparent in the patterns of mortality from certain diseases that typically are major killers of infants and children.

Model mortality patterns[9] and data from causes-of-death life tables[10] are used here to illustrate the differences that exist across societies in the distribution of child deaths by age and by major cause of death at specific phases of the mortality transition. These differences serve to highlight the importance of mortality patterns themselves in shaping the mortality-fertility relationship. For the purposes of this illustration, the critical phases of the overall mortality transition are defined as follows: (*a*) a 60 per cent probability of survival to age 20 ($_{20}P_0 = 0.60$) marks the onset of the transition; (*b*) a 75 per cent probability of survival to age 20 ($_{20}P_0 = 0.75$) marks the beginning of the second stage and (*c*) a 90 per cent probability of survival to age 20 ($_{20}P_0 = 90$) represents the end of the second stage or the beginning of the advanced stage of the transition.

As can be seen in figure II, the incidence of deaths among infants (0-1 year), children (1-5 years) and youths (5-20 years) differs markedly across the eight model mortality patterns at the onset of the mortality transition, despite the same probability of survival to age 20. At this stage, the mortality conditions implied by the East, West and Chilean model life tables were much more favourable to the adoption of conscious family planning than those underlying the North, Far Eastern and Latin American schedules, because the mortality risks were more heavily concentrated in the youngest ages making a child's survival more secure and predictable after the first few years of life.

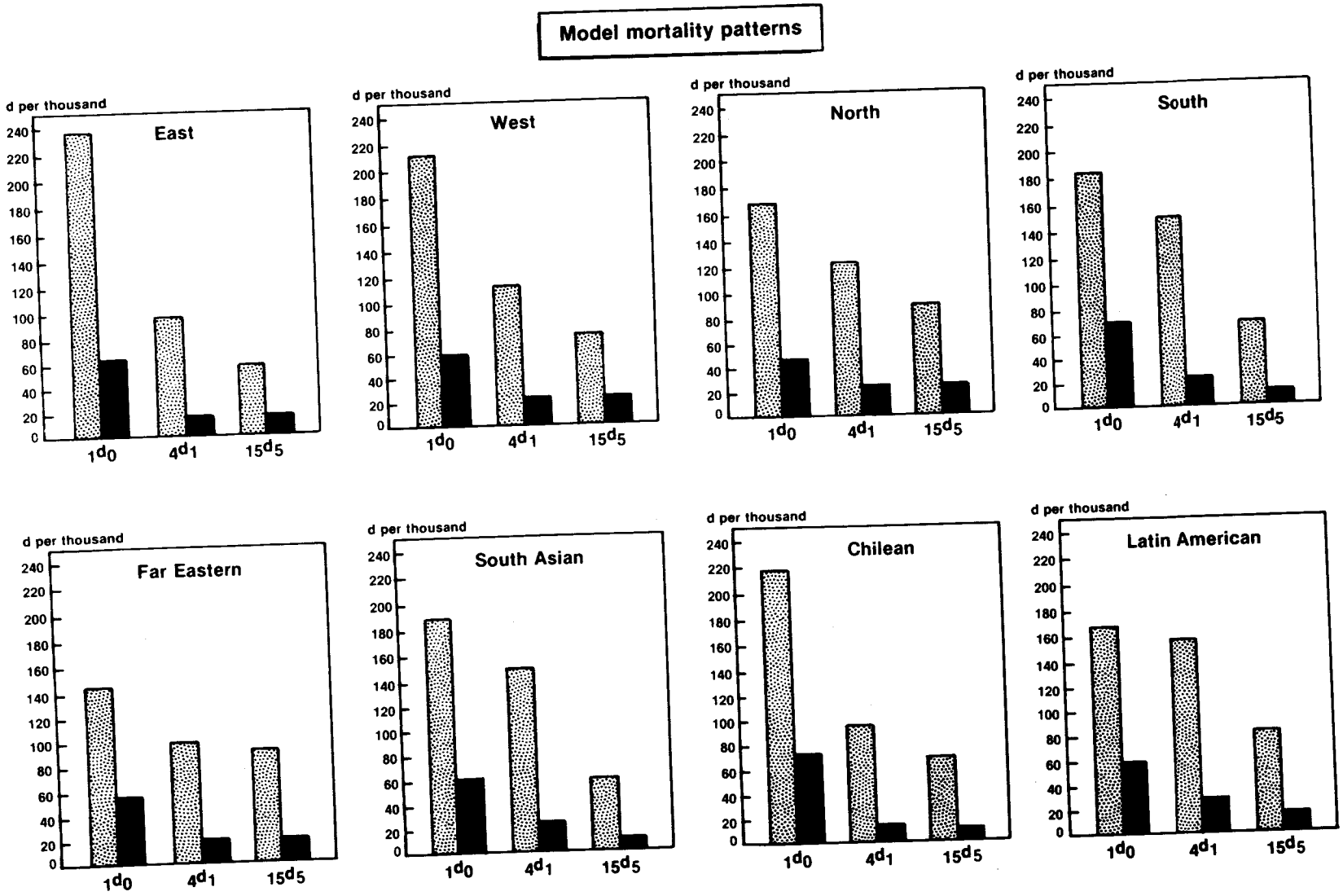
However, irrespective of the pre-transitional mortality schedule, the first stage of mortality transition appears more favourable to the initial adoption of fertility control in developing than in developed regions (United Nations, 1987a). Moreover, there is a clear relationship between the timing of the mortality transition and the direction and strength of changes in mortality schedules. The earlier the transition begins, the greater is the contribution of improvements in infant survival to overall improvements in survival. The later the mortality decline starts, the greater is the contribution of improvements in child and youth survival to overall mortality declines (United Nations, 1987a). The second stage of the transition is also more favourable to the diffusion of family planning in developing versus developed countries.

The mortality patterns implied by the South and South Asian model life tables are particularly striking because they show dramatic changes in the distribution of mortality under the age of 20 over the first two phases of the transition. At the onset of the transition, 53-54 per cent of deaths under the age of 20 occurred after the age of one in these two settings, but at the end of this process, only 32 to 39 per cent of the many fewer deaths under the age of 20 occurred over the age of one (Coale and Demeny, 1966; United Nations, 1982). Therefore, these two patterns are of particular interest because dramatic increases in life expectancy after the age of one have accompanied general improvements in child survival.

Just as the timing of the adoption of family planning and the timing and speed of its diffusion through the population depends on the age pattern of mortality, it also depends on the distribution of causes of death. While some deaths are preventable through technology transfers without any basic changes in the environment, others cannot be effectively prevented without family and community participation. Those improvements in child survival that directly involve families are likely to give them a sense of control over their fate in a way that exogenous improvements cannot. Therefore, any given improvement in mortality will be more likely to initiate fertility control behaviour among those who understand and participate in that improvement than among those who do not.

In order to analyse changes in the distribution of causes of death in the course of the mortality transition and their impact on infant, child and youth survival in

Figure II. Infant ($_1d_0$), child ($_4d_1$) and youth ($_{15}d_5$) mortality at two mortality levels ($_{20}P_0$ = 0.60 and 0.90)



NOTE: d per thousand = number dying out of a thousand at birth.

Source: Coale and Demeny (1966); United Nations (1982).

different settings, the major causes of death in the first 20 years of life are grouped into three broad categories: (*a*) "certain diseases of infancy" and diarrhoeal diseases, (*b*) respiratory diseases, including influenza, pneumonia and bronchitis; and (*c*) "other infectious and parasitic diseases" and respiratory tuberculosis.[11] Roughly speaking, only the causes of death in the third category can be efficiently attacked through technological interventions dissociated from broader social and economic changes. Inadequate diet, lack of sanitation, poor housing conditions, ignorance of personal hygiene, and lack of potable water are all conditions in which causes of death from the first two categories will flourish.

Trends in mortality within each childhood age group (infant, child and youth) for the three major groups of diseases in the first phase of the mortality transition are depicted in figure III for two developing and one developed country from different socio-cultural settings and contrasting model mortality patterns. Additionally, mortality levels at the end of this phase are shown for England and Wales, which exemplify the "West" model mortality pattern in a classic industrialist environment of the late nineteenth century, and for Guatemala in 1961, which may be typical of a recent mortality transition in the Latin American context.

Developing countries entered the first stage of the mortality transition at a much later date, when the technology necessary for preventing epidemic diseases had been developed, was widely tested and produced at relatively low cost. It can be seen that the incidence of category (*c*) (infectious and parasitic) diseases is half as great in pre-transitional Chile and the Far East than it was in pre-transitional Europe. On the contrary, in these developing country settings, respiratory diseases (category (*b*)) were a much more common cause of death than infectious and parasitic diseases. This corroborates Preston's finding (1980) that epidemic infectious and parasitic diseases (category (*c*)), with the major exception of malaria, played a fairly limited role in the mortality pattern typical of developing countries, while from one half to three quarters of the mortality decline in these countries is accounted for by a decrease in deaths from respiratory diseases (category (*b*)) and tuberculosis and malaria (category (*c*)). Particularly steep decreases in mortality from respiratory diseases in all age groups in two developing countries and its rather low levels in another developing country (see figure III, panel B) suggest the same conclusion.

Mortality patterns are strongly linked to socio-economic conditions through morbidity and recovery from diseases. As can be seen from figure III, the causes of death (category (*c*)) which are most responsive to amelioration through technology transfer without a high level of development were already playing a less important role in the probability of survival to the age of 20 in some of the developing country settings than they had in the West at the end of the first stage of the transition.[12] On the other hand, at this phase, respiratory diseases were a much more important cause of death in the developing world and one which required behavioural changes at the family level, as well as environmental changes at the community level; changes which usually accompany development.

Not only are category (*a*) diseases typically the most frequent causes of illness and death in the developing countries (Galway, Wolff and Sturgis, 1987), but the available trends show mixed records of attempts to control them (see figure III, panel A). While expanding the use of the oral rehydration therapy technique to combat dehydration caused by diarrhoea may be quite effective in particular circumstances, the main road to health in this domain lies through preventive measures. This requires safe drinking water and food, proper hygienic habits and adequate sanitation facilities—all factors that are closely linked to socio-cultural and economic factors rather than to health services.

While reductions in mortality in categories (*a*) and (*b*) are more difficult to achieve, the impact of such changes on fertility, if they can be achieved, could operate more quickly because these changes would more directly involve the family as an "agent of change". Clearly, the way in which fertility responds to improvements in child survival will depend not only on the age pattern of declines but also on changes in the distribution of causes of death.

Insurance and replacement effects

Improvements in child survival are expected to have an effect on the practice of family planning when the increased supply of surviving children that would result from such improvements exceeds parents' demand for surviving children. Under these circumstances, the availability of family-planning services would permit not only a speedier fertility response to improvements in child survival but also a more substantial one. Where family size goals are high and child survival is low, however, improvements in child survival will improve parents' chances of building the large family they so much want. In these circumstances, birth rates are not likely to fall when child survival improves, even in countries where family-planning services are readily available.

Two alternative family-building strategies have been hypothesized in the literature as ways that families, with unchanged family size goals, can make fertility adjustments to child mortality under conditions of conscious fertility control: (*a*) the "insurance strategy" and (*b*) the "replacement strategy" (Preston, 1978; Olsen, 1980; Okore, 1986). Both strategies have the same objective—to achieve the desired number of surviving children. However, they differ in a number of important ways which have implications for the speed and size of the fertility response likely to result from improvements in child mortality.

The "insurance strategy" involves the setting of a specific fertility goal in excess of desired family size in anticipation of child deaths which may occur not only during the reproductive life span but after as well, at a time when such deaths could no longer be replaced. The higher the parents' perceptions of the probability of child loss, the greater will be the excess number of births desired. In an environment of high uncertainty, a large

Figure III. Trends in mortality from specified diseases during the first stage of mortality transition[1]

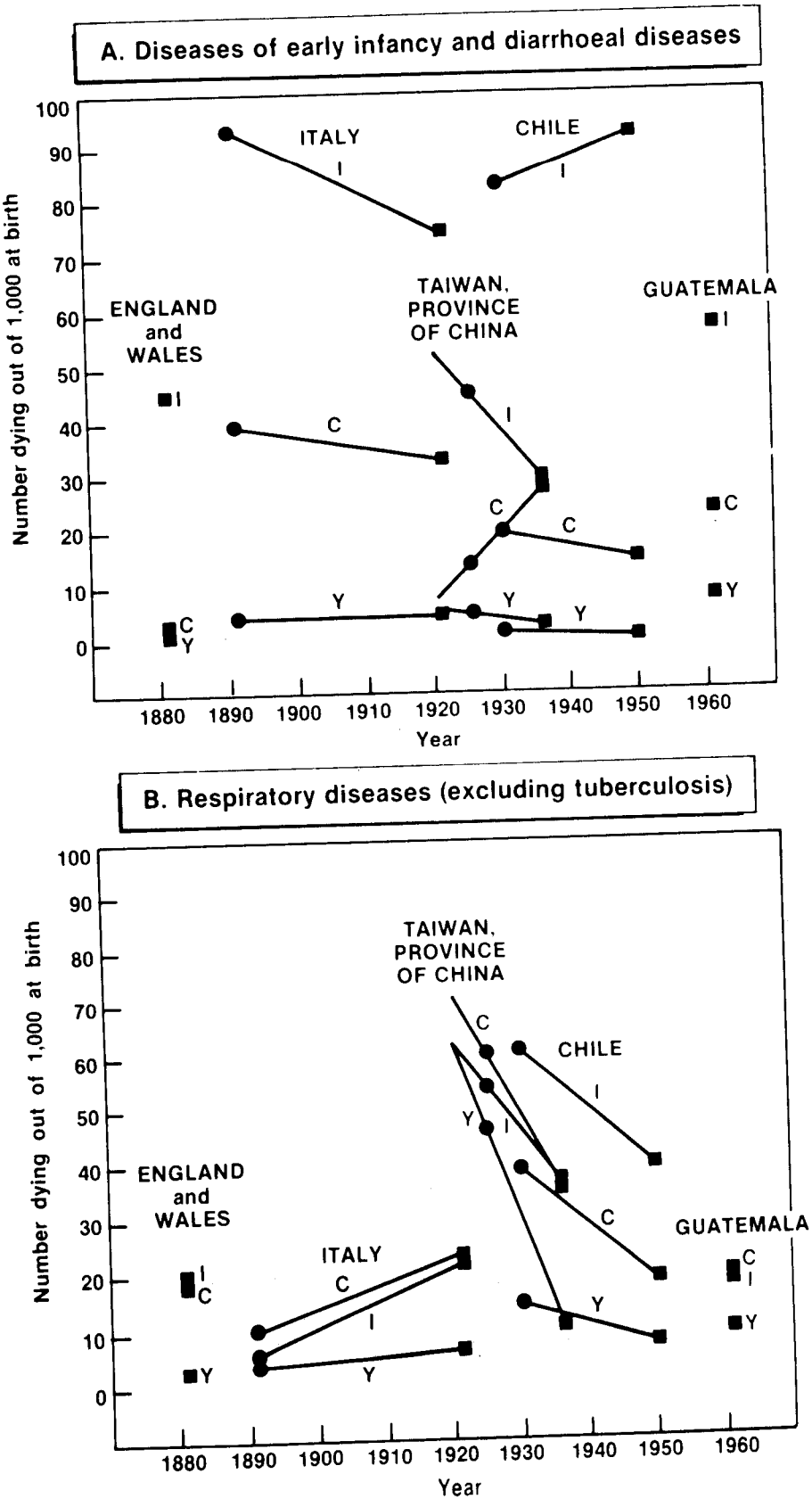
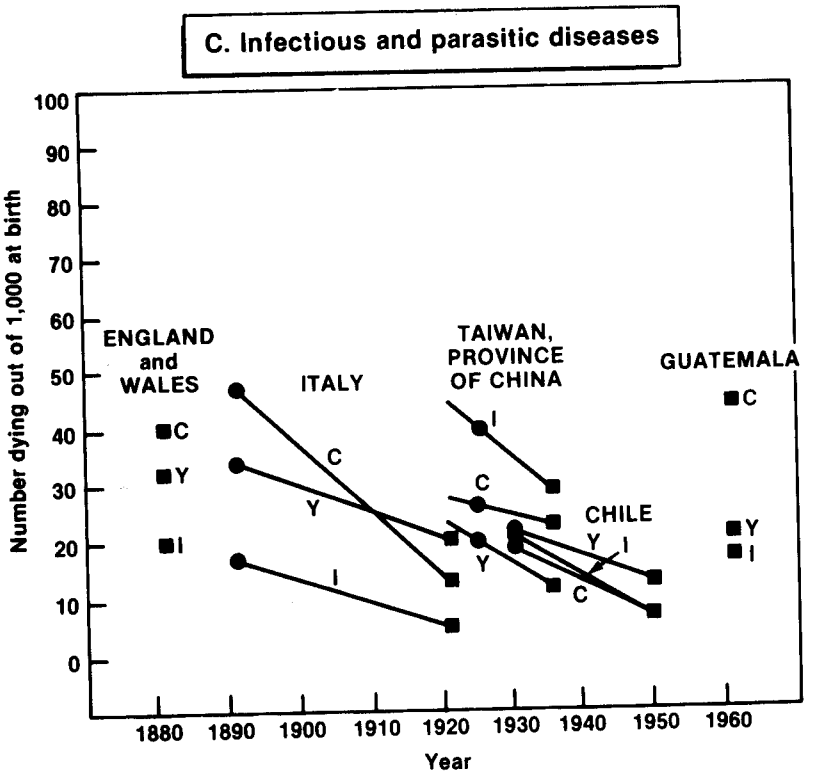


Figure III. (cont.)



Source: Preston, Keyfitz and Schoen (1972).

NOTES: The first stage of mortality transition corresponds to the increase in the probability of survival from birth to age 20 years ($_{20}P_0$) from 0.60-0.020 to 0.75-0.02.

The values of cause-specific $_nd_x$ plotted on the figure as the starting and/or final points of this stage are taken from female life tables. For Taiwan, Province of China, all graphs begin with the 1920 life table at $_{20}P_0$ = 0.54; the time when $_{20}P_0$ has reached 0.60 is roughly assessed to be at 1925.

The table below provides the dates of life tables used, their exact values of $_{20}P_0$, as well as respective pages from the Preston and others (1972) volume:

Country or region	*Date*	$_{20}P_0$	*Page*
England and Wales	1881	0.747	235
	1891	0.605	391
Italy	1921	0.743	403
	1920	0.536	703
Italy	1936	0.731	711
Taiwan, Province of China	1930	0.613	155
Taiwan, Province of China	1950	0.748	161
Chile	1961	0.735	339
Chile			
Guatemala			

proportion of parents will experience more child deaths than implied by the average level of mortality. Since parents do not know the actual probabilities of death faced by their own children, all of them are likely to behave as if they were risk averse. Therefore, the extra births needed to achieve a certain level of confidence will lead many parents to end up with more surviving children than desired.

The "replacement strategy" involves the replacement of children who die with additional birth(s) up to the end of a woman's reproductive span. This could be accomplished either by compressing inter-birth intervals, by fitting (an) extra child(ren) into the end of the childbearing life span or by a combination of both. Thus, the more widely spaced is the pattern of childbearing that parents choose, the easier it will be to replace a child death with an additional birth through a compression of inter-birth intervals. On the other hand, if parents plan closely spaced births, they will have to replace any child deaths later in a woman's reproductive span when fecundability may have declined. With the "replacement strategy", couples would practise family planning whenever they had achieved the number of living children they desired. Thus, the ready availability of efficient and reversible contraception would enhance the implementation of this strategy.

The operation of these two strategies differs in several important respects. While the "replacement strategy" requires only adaptive responses on the part of parents to actual child deaths, the "insurance strategy" requires that parents have some notion of child mortality risks, either based on their own family's or kin's past experience or their subjective impressions of the environment in which they live. These perceptions would form the basis of their fertility strategy. While the replacement strategy cannot be altered once it has been implemented, parents who choose the "insurance strategy" early in their marriage can change at a later point to the "replacement strategy" if they find that their children's survival pattern is better than anticipated.

The implications for fertility decline of improvements in child mortality are quite different in the two cases. If the "replacement strategy" is in use, an improvement in child mortality can never result in a fully compensatory reduction in births because fertility control is always imperfect. If parents replace births through a compression of inter-birth intervals, the fertility response would be fairly immediate, but the response would be lagged several years if parents wait until later in their marriage to replace child deaths. The "insurance strategy" involves an overcompensation for child deaths. Thus, an improvement in children's survival chances under this strategy has the potential for triggering a more than compensatory fertility response and, under certain conditions, even the number of surviving children and the rate of natural increase could fall.

For example, if each child faces only a 0.01 risk of dying before his/her parents die and after the point at which replacement is possible, parents who want to be 95 per cent certain that two children will survive them would need to bear only two children, as the probability that both will survive is over 0.95 (i.e. $(1 - 0.01)^2 = 0.98$). If each couple has perfect control over fertility and actually bears two children, the average number who will survive will be 1.96 (2 times 0.98). On the other hand, if mortality conditions were significantly worse, and the risk of a child's dying and not being replaced was 10 per cent, there would only be an 81 per cent chance ($(1 - 0.1) = 0.81$) that two children would survive and therefore parents would have an additional birth as insurance. With three births, there would be a 0.97 per cent chance that at least two would survive their parents (since $(1 - 0.1)^3 + 3(0.1(1 - 0.1)^2 = 0.97$). However, if all couples follow this insurance strategy effectively and each bears three births, the average number actually surviving will be 2.7. Under such hypothetical conditions, if mortality improves, the impact on fertility and ultimate family size could be quite large, even if family size desires do not change. In this case, however, the fertility response would be significantly lagged because this strategy is based on the assumption that parents act on fertility goals set at the beginning of the childbearing period.[13]

There is the possibility that parents could pursue a "mixed strategy" in order to combine the advantages of the two alternative approaches while minimizing their disadvantages. With a "mixed strategy", parents would insure only against those child deaths that might occur after the end of the wife's reproductive span but before some specified age and would replace those deaths occurring within the reproductive span only as they occurred (O'Hara, 1972; Olsen, 1980).

Insurance and replacement strategies are likely to correspond to two consecutive stages of the fertility transition. The choice between them ultimately depends on the balance between potential supply and demand for surviving children and on the type of available fertility control. The insurance strategy is more appropriate when high risks of death are spread over a long period of the child's life, when family size goals are high and/or only relatively mature children represent a net asset to their parents and/or sex preference is strong. Besides that, when only irreversible methods of family planning are available, there can be no real opportunity to pursue a replacement strategy. Finally, it has to be noted that the qualitative difference between natural fertility and insurance strategy (which requires a once and for all decision to stop childbearing) is less profound than the contrast between natural fertility and the replacement strategy (which assumes the ability to cease and resume childbearing whenever needed by effectively practising reversible birth control methods). Hence, it seems plausible to assume that the insurance strategy is most often adopted first when the idea of fertility control is relatively new.

The "replacement strategy" will ultimately take over from the "insurance strategy" at a later stage of the transition, with a "mixed strategy" sometimes providing a transition between the two. The timing of the implementation of the "replacement strategy" will depend not only on the overall course of the mortality transition but also on the incidence of deaths in the later childhood years, the availability of reversible contraceptives and the strength of son preference.

Table 3 provides a rough picture of what that transition might look like in many of the countries for which empirical findings will be reviewed below. These include all the major regions for which mortality schedules are available, as well as some additional regions that were seen to have distinctive features with respect to socio-economic conditions, as presented in table 1. It is assumed that natural fertility (N)—or family building by fate—is in use until $_{20}P_o$ reaches 0.6 and the fertility goal (B_d) is less than 9. Only when the mortality transition is under way will family building by design come into play. When the fertility goal is less than 9 and the "age of confidence" (age at which the probability of survival to the "relevant age" of 20 ($_{20}p_i$) is at least 90 per cent) is greater than 2 years, then it is assumed that the insurance strategy (I) will be adopted if contraceptive prevalence is less than 20 per cent but a mixed strategy (M) would be in place if contraceptive prevalence exceeds 20 per cent. This is because contraceptive availability provides parents more flexibility in implementing family-building strategies. For the "replacement strategy" (R) to be implemented, it is assumed for this illustration that parents must be 90 per cent confident of the child's survival to the "relevant age" before the child reaches two years[14] and contraceptive prevalence must be greater than 20 per cent.

As can be seen from table 3, these assumptions show the transition as having moved more quickly in some settings than others. In some cases the transition is further along in urban than in rural areas because of differences

TABLE 3. PROBABLE FAMILY-BUILDING STRATEGIES BY REGION OR COUNTRY, FOR SELECTED DEVELOPING COUNTRIES (1940s-1980)

Country	*1940s*	*1950s*	*1960s*	*1970s*	*1980s*
	(urban/rural)[a]				
Africa					
Northern Africa					
Egypt	N	N	I/N	M/N	R
Morocco	N	N	I/N	I/N	R/N
Sub-Saharan Africa	N	N	N	N	N
Asia					
Far East					
Republic of Korea	I	M/I	M	R	R
Taiwan, Province of China	I	M/I	R	R	R
Southeastern Asia					
Indonesia	N	N	N	M/N	M/N
Malaysia	N	N	I/N	R	R
Philippines	N	I	M/I	R	R
Thailand	N	N	M/I	M	R
Southern Asia					
Bangladesh	N	N	N	I/N	I/N
India	N	N	I/N	M/N	M
Pakistan	N	N	I/N	I/N	I/N
Sri Lanka	N	I/N	R	R	R
Nepal	N	N	N	N	N/I
Western Asia					
Turkey	N	N	I	M	R
Jordan	N	N	I	M/N	R
Latin America					
Caribbean					
Trinidad and Tobago	I	M	R	R	R
Haiti	N	N	N	I	M/I
Central America					
Costa Rica	N	M/I	R	R	R
Guatemala	N	N	I	I	R/I
Honduras	N	N	I	I	R
Mexico	N	I	M/I	R	R
El Salvador	N	I	I	R	R
Tropical South America					
Brazil	N	M/I	M/I	R	R
Colombia	N	I	M	R	R
Peru	N	N	M	R	R
Bolivia	N	N	I	I	I
Ecuador	N	I	M/I	R	R
Temperate South America	I	R	R	R	R

Sources: Family size goals (C_d) and relevant ages (j) are from United Nations (1987a), table 2. Mortality levels and timing of mortality transition are based either on actual data from the United Nations *Demographic Yearbook* (different years), country monographs from the Committee for International Co-operation in National Research in Demography (CICRED) or on estimates from United Nation (1986). Model mortality schedules are from Coale and Demeny (1966) and United Nations (1982). Causes-of-death structures of mortality transition are derived from Preston, Keyfitz and Schoen (1972). Trends in contraceptive availability are taken from United Nations (1979) and Nortman and Hofstatter (1969, 1970, 1971, 1975 and 1980) and Mauldin and Segal (1986).

NOTES: The following symbols are used:

N natural fertility
I insurance fertility strategy
M mixed fertility strategy
R replacement fertility strategy

The criteria for ascribing symbols for countries in specified time periods are:

N: If $_{20}P_0$ 0.60 or
If $_{20}P_0$ 0.60 and B_d 8
I: If $_{20}P_0$ 0.60, B_d 8, i 2 and the prevalence of contraceptive use is less than 20 per cent of women at risk
M: If $_{20}P_0$ 0.60, B_d 8, i 2 and the prevalence of contraceptive use is more than 20 percent of women at risk
R: If $_{20}P_0$ 0.60, B_d 8 and i 2

Where $_{20}P_0$ = probability of survival from birth to age 20

B_d = fertility goal (number of births desired)
i = age of confidence defined as the end of the most risky period of a child's life

The values of fertility goals (B_d) and ages of confidence (i) are assessed in a three-step procedure from countries' mortality levels (in specified periods) and model mortality schedules, assuming that family size goals (C_d) are relevant ages (j) do not change over time.

(*a*) For a given relevant age (j), mortality schedule and level, the age of confidence (i) is found such that the probability of dying in the age interval (i,j) does not exceed 10 per cent;

(*b*) The corresponding probability of survival from birth to this age of confidence ($_ip_o$) is computed;

(*c*) For this probability of survival and a given family size goal (C_d), the fertility goal (B_d, integer value) is found such that

$$\sum_{k=0}^{B_d - C_d} \frac{B_d!}{k!(B_d - k)!} (1 - {}_ip_0)^k \, {}_ip_0^{B_d - k} = 0.90$$

Appropriate family size goals and relevant ages for each setting are drawn from United Nations (1987a), table 2.

The changes in fertility strategy in response to improvements in child survival are lagged. The value of the time lag is the sum of two lag factors—the timing of the first stage of mortality transition (equals 15 years if the transition from $_{20}P_0$ = 0.60 began before 1900; 10 years if it began in 1900-1920; 5 years if it began in 1920-1950 and 0 years if it began after 1950) and its cause-of-death structure (equals 10 years if more than a quarter of the 0.15 increase in $_{20}P_0$ is associated with the decrease in mortality from infectious diseases; 5 years if this share is 15-25 per cent and 0 years if it is less than 15 per cent).

[a] Strategies are shown separately for urban and rural areas if societal conditions differ in ways that matter to the assumption of this model.

which exist in family size goals and in access to contraception. Most of the data used in the empirical studies comes from the 1960s, 1970s and 1980s. Although certain regions had already reached the point where the replacement strategy was widespread by the 1960s (e.g., East Asia and the Caribbean), most regions have undergone dramatic changes during this period (except for sub-Saharan Africa where a natural fertility régime is still in place).

In studying the empirical literature, it is important to know the decade during which data were collected in order to draw conclusions about measured relationships. When studies are based on surveys with retrospective fertility histories, births and deaths which occurred over a wide range of calendar time, possibly repeating different strategies, are often pooled to provide adequate samples for study. By the 1980s, replacement behaviour is likely to be typical in all of Asia, except Southern Asia, also in all of Latin America and in some part of Northern Africa. The notable exceptions would be sub-Saharan Africa, parts of Southern Asia, and Haiti and Bolivia in Latin America.

Much empirical research on the mortality-fertility relationship concentrates on the replacement effect because this can be tested and measured with individual survey data. Such studies relate the pace of childbearing and achieved fertility to a woman's actual experience with child death through information on the birth and child death histories of reproductive-age women. Small but significant differences in the length of inter-birth intervals between women with and without a child death have been observed in a variety of settings (Cochrane and Zachariah, 1984; DeGuzman, 1984; Rukanuddin, 1982). However, much of the replacement effect may not show up in terms of differences in length of birth interval but rather in subsequent fertility. To capture fully the replacement effect, child deaths prior to some specific parity must be related to subsequent fertility in order to avoid the confounding effects of prior fertility levels on subsequent child mortality. A common approach is to compare the number of additional children born subsequent to a specific parity to women who have experienced a child death up until that point with the number born to those who had not. Because women with a child death are likely to have had greater exposure to the risk of conception than those without, exposure must be controlled in the analysis or the measured effects will exaggerate the replacement effect.[15]

Table 4 presents selected findings from studies that have introduced appropriate controls for maternal age, exposure and socio-economic characteristics. In every case, adjusted effects are quite small, reaching 0.3 additional births per child death in the Philippines and Nepal and 0.35-0.5 in Jordan and urban Morocco. In other words, this would mean 300 fewer births for every 1,000 fewer child deaths. It has to be noted that adjusting for demographic characteristics alone considerably decreases estimates of replacement. Moreover, direct control for duration of exposure makes a greater difference in estimates of replacement than indirect controls such as the duration of marriage.

Several studies have broken the analysis down by cohort, time period of the birth or contraceptive practice so that changes over time in the strength of the response can be analysed. For the Republic of Korea, Park, Han and Choe (1979) analysed parity progression ratios for three subsamples according to whether the last child was born before 1955, from 1955 to 1964 or from 1965 to 1971. It was found that the differences in parity progression ratios between those whose previous child had survived and those whose previous child had died were negligible for previous births occurring in the first period, small in the second period and greater in the last period. This would suggest the strengthening of the replacement effect as family-planning practice becomes more widespread. Nur (1985) compared differences in mean number of additional children ever born between women with and without previous infant deaths according to whether women were contraceptive users or not. Measured replacement effects were much stronger among users than non-users and differences were statistically significant, confirming earlier hypotheses that intentional responses require that parents have control over their fertility. These results roughly suggest at least 50 per cent replacement among contraceptive users, at the higher parities.

Mensch (1985) has tried to distinguish statistically the replacement effect from the physiological effect by analysing the birth interval associated with a particular parity according to the child death experience occurring prior to the preceding parity. For example, the probability of giving birth within the five years after the third birth was related to whether or not the woman experienced the death of her first or second child.[16] Thus, the impact of child death on the post-partum infecundable period is removed from the analysis. Table 5 shows the results for Colombia, Costa Rica and the Republic of Korea for intervals occurring since 1965, comparing the behaviour of those who practise contraception with those who do not. As expected, no replacement effect is found among non-users. The replacement effect among contraceptive users is apparent in the third birth interval for Costa Rica, the fourth birth interval for the Republic of Korea and not at all in Colombia. This is roughly consistent with the family-building strategies depicted for these three countries in table 3, with Costa Rica showing replacement behaviour even in rural areas in the 1960s, the Republic of Korea showing a mixed strategy and Colombia showing the insurance strategy. As replacement behaviour is unlikely to affect the pace of childbearing until the desired number of children have been born, it is not surprising that the behaviour is manifested at an earlier parity in Costa Rica than in the Republic of Korea because, between 1965 and 1975, Costa Rica showed a much more rapid fertility decline and had an average total fertility rate of 3.8 at the end of the period, in contrast to 4.3 for the Republic of Korea (United Nations, 1983).

Several studies have looked directly at contraceptive use in relation to child survival (Rutstein and Medica, 1975; Ware, 1976; Uyanga, 1982; Cochrane and Zachariah, 1984; De Guzman, 1984; Ebanks, 1985;

TABLE 4. REPLACEMENT RESPONSE TO THE DEATH OF ONE OR TWO OF THE FIRST FEW CHILDREN

Parity (i)	Country, area	Excess mean number of subsequent live births over the reference group[a] if: One of the first i children died — Unadjusted	One of the first i children died — Adjusted for major demographic factors[b]	One of the first i children died — Adjusted for all factors[c]	Two of the first i children died — Unadjusted	Two of the first i children died — Adjusted for major demographic factors[b]	Two of the first i children died — Adjusted for all factors[c]
1	Costa Rica, rural	1.0	0.2	0.1			
	Colombia, rural	0.7	−0.1	0.0			
	Mexico, rural	0.7	0.0	0.0			
	Peru, rural	2.3	0.5	0.35			
	Nepal	. .	. .	0.3			
	Philippines	0.9	0.4	0.3			
	Jordan	1.4	0.45	0.35			
	Pakistan, urban	1.5	0.6	0.4			
	Pakistan, rural	0.8	0.4	0.1			
	Sri Lanka, urban	0.4	0.2	−0.1			
	Sri Lanka, rural	0.8	0.3	−0.1			
2	Costa Rica, rural	1.1	0.1	0.0	2.4	0.5	0.8
	Colombia, rural	0.7	0.0	0.0	1.6	0.1	0.0
	Mexico, rural	0.8	0.0	0.0	1.1	−0.5	−0.5
	Peru, rural	0.8	0.2	0.1	1.1	0.1	0.1
	Nepal	. .	. .	0.3	. .	. .	0.4
	Philippines	0.7	0.25	0.2	0.8	0.5	0.5
	Jordan	1.3	0.5	0.5	2.4	1.0	0.9
	Pakistan, urban	0.7	0.3	0.0	1.7	0.8	0.4
	Pakistan, rural	0.8	0.4	0.2	1.3	0.6	0.2
	Sri Lanka, urban	0.3	0.2	0.1	1.6	0.8	0.0
	Sri Lanka, rural	0.6	0.3	0.1	0.95	0.2	−0.3
3	Costa Rica, rural	1.2	0.4	0.2	2.3	0.2	0.0
	Colombia, rural	0.7	0.1	0.1	1.0	−0.2	−0.2
	Mexico, rural	0.8	0.1	0.1	1.5	0.3	0.35
	Peru, rural	0.8	0.2	0.15	0.9	0.1	0.05
	Nepal	. .	. .	0.2	. .	. .	0.3
	Philippines	0.4	0.2	0.1	1.0	0.3	0.3
	Jordan	1.1	0.4	0.4	1.8	0.6	0.5
	Taiwan, Province of China, rural	1.5	. .	0.3	2.15	. .	0.1
	Pakistan, urban	0.9	0.55	0.25	1.65	0.85	0.3
	Pakistan, rural	0.7	0.3	0.15	1.2	0.6	0.1
	Sri Lanka, urban	0.5	0.5	0.3	0.2	−0.6	−0.9
	Sri Lanka, rural	0.4	0.2	0.0	1.0	0.5	0.0
	Morocco, urban	0.7	. .	0.6	1.0	. .	0.7

Source: Costa Rica, Colombia, Mexico and Peru: Balakrishnan (1978), table 5; Nepal: Chandran (1981), table 1-3; Philippines: De Guzman (1984), table 10.5; Jordan: Nur (1985), table 1; Taiwan and Morocco: Heer and Wu (1975), tables 1 and 2; Pakistan and Sri Lanka: Rao and Beaujot (1986), table 3.

NOTE: Two dots (. .) indicate that data are not available.

[a] Mean number of subsequent births for women whose first (i) children survived.

[b] Adjusted for age and elapsed duration since the last birth for Costa Rica, Colombia, Mexico, Peru, Nepal, Philippines and Jordan; age and duration of marriage for Pakistan, Sri Lanka and Morocco.

[c] For Costa Rica, Colombia, Mexico and Peru, adjusted for age, elapsed duration, ideal number of children, occupation of husband, standard of living, education of wife, place of residence, church attendance, and perception of child mortality; for Nepal, adjusted for age, elapsed duration, duration of breast-feeding, and contraceptive use; for the Philippines, adjusted for age, elapsed duration, occupation of husband, education and work status of wife and place of residence; for Jordan, adjusted for age, elapsed duration, education of wife, her childhood place of residence, occupation of husband; for Taiwan, adjusted for age, elapsed duration, duration of breast-feeding, knowledge of contraception, education and work status of wife, occupation and income of husband, perception of child survival and preference for sons; for Pakistan, adjusted for age, duration of marriage, ideal number of children, mean birth interval, education of wife, her occupation before first marriage, duration of breast-feeding, education and occupation of husband; for Sri Lanka, adjusted for age, duration of marriage, ideal number of children, mean birth interval, education of wife, her occupation before marriage, religion affiliation, standard of living, household structure; for Morocco, adjusted for age, duration of marriage, age at first marriage, literacy, work status and birth place of wife, literacy, occupation, income and birth place of husband.

TABLE 5. RELATIVE RISK OF ADDITIONAL BIRTH FOR WOMEN WITH CHILD DEATH EXPERIENCE[a]

Country	Non-contraceptors, parity (i)				Contraceptors, parity (i)			
	. .	0.8	1.0	1.2	. .	0.9	0.7	1.2
Colombia	0.9	1.0	0.8	. .	2.8	0.9	1.0	. .
Costa Rica	0.9	1.0	1.0	. .	1.7	2.5	1.6	. .
Republic of Korea								

Source: Mensch (1985), tables 4-6.

NOTE: Two dots (. .) indicate that data are not available.

[a] Ratio of the probability of ((i) + 1)th birth within five years since the (i)th birth for women who have lost at least one of their first ((i) − 1) children to the similar probability for women whose ((i) − 1) children survived. Adjusted for age, education, place of residence and year of the (i)th birth.

Mensch, 1985; United Nations, ESCAP, 1985). In general, results confirm that better child survival is associated with higher levels of contraceptive use but only in those countries where fertility control is widespread. In table 6, results from several World Fertility Surveys are compared and it is particularly striking to note that in Indonesia, where child death rates are much higher than in the Philippines, Thailand, Sri Lanka, Republic of Korea, Fiji and Malaysia, contraceptive use differentials between women with and without a child death experience are greater than in these six other countries of the region. These results suggest that behavioural responses can be strengthened even in an unfavourable mortality environment through the widespread distribution of contraception. Mensch's (1985) results support the view that contraceptive behaviour should show its strongest response to child deaths during those intervals that correspond most closely to the achievement of desired family size. Thus, the strongest differences in contraceptive behaviour are found among women at the lower parities in settings where the demographic transition is relatively far along and at the higher parities where it has not progressed as far.

The few empirical studies which have been designed specifically to detect insurance effects do indeed demonstrate the tendency for families to adjust their childbearing practices and attitudes to the mortality conditions they face. The technique is to regress actual or intended fertility behaviour either on subjective parental perceptions of child survivorship or on some objective measure of current mortality conditions. Thus, Heer and Wu (1975) found that in Taiwan, Province of China, the perception of children's better survival chances is associated with substantially lower fertility; residence in a township with lower infant and child mortality is associated with lower fertility (after holding constant major variables at the individual level). The study by Rizk, Stokes and Nelson (1980) in Egypt shows that the proportion of women practising contraception, adjusted for individual-level variables (including child loss) and the distance of the village from family-planning services, is much higher in villages with relatively low infant mortality. The study by Pebley, Delgado and Brinemann (1979) in Guatemala leads to the conclusion that, while the subjective perception of child survival does not have any significant effect on the desire for additional births, the proportion of respondent's siblings who had died in infancy or childhood is positively related to fertility preferences. In order to detect the impact of the level of infant and child mortality on fertility behaviour and attitudes independent of the individual experience of child loss, Heer and Rodriguez (1986) combined the information for individual women from the 1976 World Fertility Survey in Costa Rica with aggregate statistics for 104 community areas obtained in the 1973 census. Their results suggest the absence of such an impact, which is consistent with the prolonged prevalence of replacement strategy (and, hence, the weakness of the insurance elements in the fertility behaviour) in this fairly advanced developing country (see table 3).

All these studies suggest that the insurance effect is far less than compensatory. However, these results may be due to measurement problems in some cases and/or the

TABLE 6. EFFECT OF CHILD DEATH EXPERIENCE ON CURRENT CONTRACEPTIVE PRACTICE

Country	Per cent of children dying before age 5	Per cent currently using contraception among exposed women wanting no more children	Contraceptive use differentials by child death experience: Percentage point difference in contraceptive use between women with and without child death experience[a]	Contraceptive use differentials by child death experience: Percentage point reduction in contraceptive use owing to one child death[b]
	1	2	3	4
Nepal	23	9	2	2
Bangladesh	22	20	2	2
Pakistan	21	15	3.5	1.5
Indonesia	16	53	10	7.5
Philippines	9	60	8	5
Thailand	9	56	9	. .
Sri Lanka	9	54	6	5
Republic of Korea	6	56	8	6
Fiji	6	69	8.5	6
Malaysia	5	53	6	6

Sources: Column 1: Rutstein (1984), table 3; column 2: United Nations (1987b), table 79; column 3: United Nations, ESCAP (1985), table 4; column 4: Cochrane and Zachariah (1984), table 5.

NOTE: Two dots (. .) indicate that data are not available.

[a] Adjusted for number of children ever born, wife's age and the square of her age, education and place of residence and husband's occupation and education.

[b] Adjusted for number of children ever born, wife's place of residence and work status and husband's occupation.

choice of inappropriate settings rather than to the intrinsic weakness of this effect in those settings where it is in practice. In fact, the reliability of attitudinal questions is fairly limited in general; the answers to the questions which address such a sensitive issue as the anticipated deaths of the respondent's own children are likely to be particularly affected by cultural taboos, psychological reluctance to discuss intimate fears with a stranger and uneasiness to give numerical responses. Alternatively, the use of a more "objective approach" to the measurement of perceptions implies the regression of data from individual fertility histories on the level of child survival in the community (assumed to reflect perceptions). This in turn, is often precluded by the practical impossibility of isolating the influence of the level of infant and child mortality from the influence of other relevant features in the environment, or, in other words, of finding two communities with substantially different mortality conditions but identical social, economic and cultural characteristics.

Additionally, the use of current community levels of infant and child mortality as the proxies for the determinants of the "insurance effect" may be misleading, especially if the respective communities underwent substantial and unequal improvements in child survival in the recent past. Finally, the results may be biased if cohorts of women with presumably different types of fertility behaviour are pooled together. Thus, a potentially more promising approach could be to relate fertility behaviour of distinct cohorts of women with mortality conditions prevailing at the time of their childhood or entry into marital unions, as well as with the magnitude and pace of improvements in children's survival chances achieved since this period. This approach may provide a better opportunity to detect more accurately (at least for relatively older cohorts with near completed fertility) differentials in "excess fertility" conditioned by differences in mortality conditions across communities, as well as to test the hypothesis of the depletion of insurance motivation in a longitudinal perspective. However, such a study is difficult to implement in practice because of its data requirements.

The decline in the prevalence of the insurance motivation in settings where mortality has fallen steeply may be inferred from the greater tendency of women in such settings to make up the loss of a child (that is to practise "replacement strategy"), which is not necessary if one insures against the loss in advance (Schultz, 1978; Ben-Porath, 1976; Schmitz, 1985). However, this does not by itself help in assessing the actual impact of mortality on fertility when the insurance strategy is practised.

Because of the difficulty of measuring the macro-demographic effects of improved child survival on fertility in settings where the "insurance strategy" is in place, simulations have been used to quantify the range of plausible effects. Using information on the pattern and pace of mortality decline in different settings (see section on the transition effect), as well as certain notions about the likely family size goals and "relevant ages" characteristic of different socio-economic settings (from table 1) the fertility decreases that might be attributable to the "insurance effect" during the first stage of the mortality transition ($_{20}P_0$ from 0.60 to 0.75) are simulated (see table 7). The advantage of this approach is that it shows how different mortality and socio-economic environments could have an impact on the extent and the timing of the fertility response to mortality decline. This simulation assumes that parents have perfect control over fertility and choose the fertility goal (B_d) once and for all at the time they first enter marriage. In addition, it is assumed that changes in mortality are perceived without a lag and result in an immediate fertility response. The fertility goal chosen by parents at their entry into marriage will be that number sufficient to assure them, with a confidence level of 90 per cent, that their desired number of children will survive to the "age of confidence"—the age at which they have at least a 90 per cent probability of reaching the "relevant age".

In table 7, the fertility goals under the insurance strategy are seen to vary according to the prevailing mortality pattern, the "relevant age" and family size goals. Looking at a "relevant age" of 20, which has been assumed to be typical in most developing country settings, it can be seen that, at the start of the transition, slightly more births would be required to protect against child losses under the Latin American and South Asian mortality schedules than under the Far Eastern and West schedules. A very high "relevant age" of 30 would require additional births in all settings. The higher the family size goal, the greater the impact of the "relevant age" on the fertility goal. In the course of the first stage of the transition, the fertility goals decrease in all settings for family size goals greater than one. In those settings where mortality among youth remains relatively high, such as those with the West and Far Eastern mortality patterns, the appropriate "relevant age" makes a big difference in the number of births required to compensate for child losses. For example, at the end of the first stage of the transition, in the Far Eastern pattern at a family size goal of four, one additional birth would be required if the "relevant age" were 15, two additional births if the "relevant age" were 20 and three if the "relevant age" were 30. In the Far East, where son preference is strong and the "relevant age" is likely to be high, the first stage of the transition would yield a large decline in birth rates and the largest percentage decline in intergeneration growth because this is the setting where the insurance strategy required the greatest number of compensatory births.

Table 7 demonstrates clearly that improvements in child survival at the early stages of the demographic transition can generate declines in birth rates and growth rates if the changes are fully perceived by parents and if they can exercise fairly effective control over fertility. The largest declines should occur in those settings where improvements in child and youth mortality have been particularly strong and where mortality patterns were originally relatively unfavourable during the childhood years.

Demand effects

In the previous section, attention was given to the plausible effects of actual or anticipated child survivorship on the number of births needed to achieve a particular family size goal. In other words, it was implicitly

TABLE 7. INSURANCE STRATEGY: SIMULATED DECREASES IN FERTILITY GOALS AND CHANGES IN NUMBERS OF SURVIVING CHILDREN GENERATED BY THE FIRST STAGE OF MORTALITY TRANSITION

Relevant age j	Family size goal C_d	Mortality pattern based on model life tables: West — Fertility goal at $e_0 = 35$ (1)	West — B_d at $e_0 = 45$ (2)	West — Change in survey (3)	Latin America — Fertility goal at $e_0 = 35$ (4)	Latin America — B_d at $e_0 = 45$ (5)	Latin America — Change in survey (6)	South Asian — Fertility goal at $e_0 = 35$ (7)	South Asian — B_d at $e_0 = 45$ (8)	South Asian — Change in survey (9)	Far Eastern — Fertility goal at $e_0 = 35$ (10)	Far Eastern — B_d at $e_0 = 45$ (11)	Far Eastern — Change in survey (12)
15	1	2	2	+0.30	2	2	+0.30	3	2	−0.30	2	2	+0.20
	2	4	3	−0.15	4	3	−0.15	5	4	0.0	4	3	−0.35
	3	6	5	+0.15	6	5	+0.15	7	5	−0.45	5	4	−0.25
	4	8	6	−0.30	8	6	−0.30	9	7	−0.15	7	5	−0.80
20	1	3	2	−0.30	3	2	−0.30	3	2	−0.30	2	2	−0.20
	2	4	3	−0.15	5	3	−0.75	5	4	0.0	4	3	−0.35
	3	6	5	+0.15	7	5	−0.45	7	5	−0.45	6	4	−0.90
	4	8	6	−0.30	9	6	−0.90	9	7	−0.15	8	6	−0.70
30	1	3	3	+0.45	3	2	−0.30	3	2	−0.30	3	2	−0.45
	2	6	4	−0.60	6	4	−0.60	5	4	0.0	5	4	−0.25
	3	8	6	−0.30	8	6	−0.30	8	6	−0.30	8	6	−0.70
	4	10	8	0.0	10	7	−0.75	10	7	−0.75	10	7	−1.25

Source: For mortality schedules, see Coale and Demeny (1966) and United Nations (1982).

NOTES:

Col. 3 (or 6 or 9) = col. 1 (or 4 or 7) × 0.60 − col. 2 (or 5 or 8) × 0.75

Col. 12 = col. 10 × 0.65 − col. 11 × 0.75 because in the Far Eastern pattern,

$e_0 = 35$ corresponds to ${}_{20}P_0 = 0.65$ and $e_0 = 45$ corresponds to ${}_{20}P_0 = 0.75$.

For a given j, C_d, e_0 and model mortality schedule, B_d is derived from the model presented in the footnote to table 3.

assumed that the desired number of surviving children itself was not affected by the level and pattern of mortality. This assumption may not always be plausible. Indeed, family size goals and, consequently, fertility plans may sometimes change according to a couple's own experience with child death. For instance, couples who had anticipated some child losses may revise downward their family size goals if the actual number of child deaths exceeds the anticipated number (Namboodiri, 1983; Schultz, 1978). On the other hand, a much better than expected experience with child survival may induce couples to proceed to higher parities because the process of childbearing and rearing was less costly than expected. From generation to generation, family size norms and preferences could rise as family building becomes a less risky undertaking in the course of the mortality transition. The general case of the interplay of the effects of child survival on the demand for children and the demand for births has been described by Schultz (1976) as follows:

> "If we assume that parents want children in order to obtain economic and other benefits, infant and child mortality exert two offsetting effects on fertility. First, a reduction in child mortality increases the number of survivors demanded by decreasing the expected 'cost' (including monetary, opportunity, and psychic costs) to parents of bearing and rearing enough offspring to obtain a survivor. Second, it decreases the derived demand for births for a fixed surviving family-size goal of parents by decreasing the number of births required to obtain, on average, a survivor. The former price effect of decreased child mortality on demand should induce parents to want more surviving offspring, but fewer births will be required for any desired number of surviving children, because of the latter supply effect."

These two effects—the price and the supply effect—work in opposite directions and their net effect on the desired number of births is likely to vary according to socio-economic setting. Schultz's "supply effect" is equivalent to the insurance and replacement strategies previously discussed. The "price effect" will moderate the effect on fertility of any given improvement in child survival as discussed earlier, thus slowing the fertility transition. It is likely to have its largest effects in settings where mortality is high and the value of surviving children remains high even at relatively high parities. This is most likely to be the case in rural settings where family ties are strong, child labour is highly valued and land is relatively abundant or migration opportunities are promising, as is the case in sub-Saharan Africa and in some parts of Asia. In addition to the "price effect" of improved child survival, the values parents derive from children could also improve because improved health among children could raise their labour value in settings where child labour is important and child health is generally sufficiently poor that it would otherwise limit their labour contribution. Under these circumstances, there is the possibility that improvements in child survival could lead on balance to an increased demand for births.

More typically, however, the value of incremental surviving children diminishes at higher parities because needs for risk insurance diminish after family size reaches some critical level and child labour reaches diminishing returns. Therefore, the "price effect" may have a limited impact when actual family size is already within this critical range.

Potentially, a more important effect of improvements in child survival than the "price effect" over the long term is the "quality effect", which occurs if parents are induced to invest more in quality per child, while reducing the desired number of children (O'Hara, 1972, 1975; Ben-Porath, 1976). In other words, when parents become more confident that their children will live and, thus, become more assured of the returns for physical and emotional investments in their children, they may become more inclined to devote scarce resources to child rearing. Since resources remain limited, the corresponding constraints would condition the move from large quantities of children towards fewer children of higher quality (Becker and Lewis, 1973; Willis, 1973; Becker, 1981). Thus, this "quality effect" is likely to result in fewer children being desired in a régime of low mortality. Moreover, from the intergenerational perspective, increases in investments per child would affect children's fertility behaviour in adulthood, with fertility preferences declining with the rising aspirations of younger generations.

The possibilities for declines in fertility preferences in response to mortality improvement should be greatest in countries in which parents find many opportunities to invest meaningfully in their children. The greater the quality of schools and the greater is the perception that education produces rewards in the future, the greater should be parents' incentives to trade off quantity for quality as mortality falls (Schmitz, 1985).

It has to be noted that, from the analytical viewpoint, it is impossible to distinguish statistically among insurance behaviour, "price effects" and quantity-quality interactions. Hence, when all these three mechanisms act in the same direction (or at least when the positive "price effect" is weak), which is highly likely to occur now in most socio-cultural settings of the developing countries, the fertility-depressing effects of improvements in child survival would inevitably be underestimated by studies based on individual data because some of the effects of improvements in child survival operate outside the individual family's experience with child deaths.

Policy implications

Theoretical considerations, largely supported by empirical research findings, suggest that levels and trends in child survival are negatively associated with levels and trends in fertility but the underlying mechanisms are not identical in different populations. The actual fertility consequences of a particular health intervention depend not only on the type of intervention but also on the prevalent family-building strategy and the nature and scope of family-planning programmes in the particular location in question.

In natural fertility settings where customs dictate prolonged breast-feeding, the physiological mechanism linking infant deaths and birth interval length is at its strongest. Here, a health intervention requiring little active involvement of the targeted population, such as a large-scale immunization programme, can be effectively used to derive the maximum fertility benefit from the non-volitional "physiological effect". However, the weakening of breast-feeding practice in such circumstances gravely threatens the beneficial effects of such campaigns: on the health side by increasing the exposure of infants to diarrhoeal diseases and on the fertility side by weakening the link between a child's survival and the length of the birth interval. In Africa, where far less than a third of the children are fully immunized by age one against six major communicable diseases (Sherris and others, 1986), immunization programmes should be closely integrated with educational programmes explaining the health benefits of breast-feeding.

Unlike the "physiological effect", other fertility-depressing effects of improvements in child survival involve intrinsic changes in the family-building process and only become apparent at subsequent stages of the mortality transition. The potentially most important of these but the least subject to empirical testing is the "transition effect" through which improvements in child survival, which increase the predictability of the family-building process, trigger the transition from natural to controlled fertility behaviour. This, in turn, generates the need for family planning. Until this "transition effect" occurs, the implementation of family-planning programmes cannot precipitate significant changes in fertility behaviour and, thus, cannot play an important role in the improvement of child health.

The timing of the transition to deliberate fertility control depends on the age distribution of mortality improvements as well as on the sources of improvements in terms of causes of death. The growing need for broadly defined "social" approaches to public health becomes evident from an examination of existing patterns of causes of death in the developing countries, where diarrhoeal and respiratory diseases are major killers of infants and children. While decisive reductions in the incidence of these diseases are more difficult to achieve than reductions in the incidence of infectious diseases, their ultimate impact on fertility behaviour should be faster and more profound. Female education emerges as an important intervening factor not only in translating health policies into effective tools for the achievement of improvements in child survival but also in translating those improvements into reductions in birth rates.

When parents are able to adjust their childbearing plans to the child survival conditions they face, this adjustment may be accomplished either through "insurance" or "replacement" strategies. These two adjustment strategies correspond to two consecutive stages of the fertility transition. The "insurance strategy", which is easier for formerly natural fertility populations to adopt, is fairly adequate when considerable risks of death are spread over a long period of a child's life

and when the practice of parity-specific fertility control is beginning. The "replacement strategy" becomes appropriate and feasible at a later stage when child survival is relatively certain after some fairly young age and when parents are able to cease and resume childbearing whenever needed by effectively using reversible birth control methods. Thus, in order to enhance the fertility-depressing effects of child survival improvements, programme managers must vary the mix of programmatic components in an integrated child health/family planning policy according to local circumstances.

For example, in Pakistan and Bangladesh in Asia and Haiti and Bolivia in Latin America, child survival rates are still too low for families to practise a replacement strategy. In these circumstances, the promotion of sterilization to assist families in the termination of childbearing when desired fertility has been achieved would be more realistic as a first step and would take advantage of the fertility-depressive potential of the "insurance strategy". Then, when child survivorship has improved sufficiently, the emphasis can shift to reversible contraception. India is likely to be approaching such a threshold: the formerly appropriate emphasis on sterilization will, if unchanged, hamper the adoption of the replacement strategy as child survival improves.

Countries currently characterized by moderately high infant mortality and rather low child mortality have particularly promising prospects for the quick translation of further improvements in child survival into lower fertility if there is a diversity of accessible options for family planning available. While this is the case of Brazil, Colombia, Ecuador, El Salvador and Mexico, quite similar mortality conditions in Algeria, Jordan and Turkey as well as Guatemala, Honduras and the Philippines, are not matched by the adequate accessibility of modern reversible contraceptives. Thus, in these countries, efforts to depress infant mortality might be combined with a family-planning policy emphasizing reversible contraception.

On the other hand, in Egypt and Peru, and, to a lesser extent, in Indonesia, the availability of reversible contraceptives appears greater than the current level of child survival might seem to warrant. In these countries, first priority should be given to health measures in which parents themselves can be involved and which can induce a strong response in terms of improved family-planning adoption. The availability of sterilization would also provide a welcome option for couples using the insurance strategy who need effective means of terminating childbearing. In fact, it would seem that there would be good opportunities to intensify the impact of improvements in survival on fertility through the universalization of formal education of children, educational and informational campaigns aimed at compressing the gap between the actual improvements in child survivorship and its perception at communal/individual levels, integration of child health programmes within family planning programmes, and easy access to safe, efficient and reversible methods of family planning. The proper identification of each setting in terms of transition stage and likely patterns of family building is necessary for the design and interpretation of research findings, as well as for the implementation of child health interventions which minimize consequent short-run increases in population growth rates. The importance of integrating family planning with health care delivery is strongly implied by these results.

NOTES

[1] The World Population Plan of Action includes reduction of infant and child mortality among the six socio-economic development goals that tend to moderate fertility (United Nations, 1975, chap. I, para. 32). The International Conference on Population, in its recommendation 35, reiterated improved health as a means to decrease fertility (United Nations, 1984, chap. I, para. 26). According to the World Health Organization, "where infant and childhood mortality are high, isolated family planning programmes are unlikely to convince unresponsive or resistant couples" (World Health Organization, 1975, p. 466). According to the United Nations Fund for Population Activities (UNFPA), birth rates can be expected to remain high in countries where infant mortality rates are high (United Nations Fund for Population Activities, 1984). Thus, UNFPA considers the issues of infant mortality to be closely related to its activities in the family-planning field (United Nations, Economic and Social Council, 1987, para. 22). The United Nations Children's Fund maintains that child survival tends to reduce birth rates and to slow population growth (United Nations Children's Fund, 1984, p. 32; 1985, p. 22; 1986, p. 78; 1987, p. 10).

[2] For the full report of the study, see United Nations, 1987a. Highlights from it were presented in a background paper, entitled "The effects of improved child survival on family planning practice and fertility", at the International Conference on Better Health for Women and Children through Family Planning (Nairobi, 1987).

[3] The labour-intensive agriculture prevailing in sub-Saharan Africa and still persisting in Asia favours the use of child labour much more than the capital-intensive agriculture of Europe and Latin America. With respect to land tenure systems, the communal and para-feudal land ownership system in pre-industrial Eastern Europe produced land allocation rules that favoured households with more children, particularly boys. In Latin America, the large majority of peasants lease small plots of land (minifundio) from the owner of the large estate (latifundio) in exchange for (child) labour supplied to the latifundio owner; but with subdivision of land and the mechanization of agriculture on large estates, the labour value of children is reduced. In Africa, the abundance of land in certain settings had permitted communal ownership and shifting cultivation, allowing parents to increase land under cultivation through child labour with no negative consequences in terms of productivity or the division of the land. However, in many parts of Africa today widespread soil erosion, deforestation and desertification have decreased the carrying capacity of the land and undermined the communal land tenure system, thus reducing the value of additional child labourers. In Asia, where land is scarce and highly valued by families, the input of additional child labour will run into diminishing returns, but an appropriate strategy for increasing economic security may be to divide family incomes between those derived from agriculture and those derived from non-farming activities, leading to high family size goals.

[4] The family structure is a particularly strong correlate of the demand for children. Thus, a family system based on lineage with strong kinship ties—as in some sub-Saharan societies—lessens the need of individual parents for the support of their own children. The joint family system which prevails today in most Asian and Latin American countries is the structure where the need for such support may be the greatest. The family structure has implications for the costs of children as well. For instance, the child-fostering system, which is deeply ingrained in the texture of some sub-Saharan rural societies, substantially moderates the impact of ongoing modernization on the costs of children to their natural parents.

[5] Thus, some cultures (especially in East Asia) emphasize the obligation to assure the continuation of the family line by having a son who will outlive his parents, while others (for example, sub-Saharan tradi-

tional societies) equate numerous offspring with the fulfilment of social and religious duties.

[6] For example, in certain Moslem settings, women cannot work in the fields, thus raising the value of children as substitutes for their mothers in the field, while in settings that encourage women to participate actively in economic activities outside their households (as in Thailand), the opportunity costs of children are likely to be especially high.

[7] The elasticity is defined as the percentage change in children ever born in response to a 1 per cent change in the reciprocal of the survival probability.

[8] For instance, it was found that in the rural Khanna area of Punjab, India, the death rate of children was 50 per cent higher among females than among males (Wyon and Gordon, 1971). This death rate was measured as a ratio of the number of deaths occurring in a year in the youngest five-year age group to the mid-year estimate of the population aged 0-5.

[9] In the analysis of almost 200 empirical life tables recorded in developed societies over the past 150 years, Coale and Demeny (1966) identified four distinct patterns of mortality corresponding to geographical areas of Europe: North, West (which encompassed overseas regions of European settlements as well as Japan), East and South patterns. Some time later, the United Nations (1982) published a set of model life tables based on 72 empirical life tables for 22 less developed countries. Four additional distinct patterns were, thus, identified, which, because of the predominance of these patterns in certain geographic regions, are also identified in regional terms: Latin American, Chilean, South Asian and Far Eastern patterns. Unfortunately, no patterns from sub-Saharan Africa were available. Besides that, certain regions are not well represented in these model life tables because underlying national tables were unavailable or unreliable. These underrepresented regions include most of Asia (with the exception of the Far East) and Northern Africa. Some Asian countries—the Philippines, Sri Lanka and Thailand—were included in the Latin American model pattern.

[10] Causes-of-death life tables, which have been compiled by Preston, Keyfitz and Schoen (1972), can be linked with the model mortality schedules discussed above for at least two points in the mortality transition. Unfortunately, however, only 31 female populations (representing 12 countries), out of 180 populations (representing 48 countries) for which causes-of-death life tables are available, have life tables that characterize the first phase of the mortality transition (i.e., $_{20}P_0$ ranging from 0.60 to 0.75), and these only cover five of the eight model schedules discussed above: West, South, Latin American, Chilean and Far Eastern. These 12 countries include: (*a*) from the West mortality pattern: (i) England and Wales (five life tables for the period from 1861 to 1901; the corresponding $_{20}P_0$ range from 0.68 to 0.75), (ii) United States (1900 life table for "registration States", with $_{20}P_0 = 0.76$), and (iii) Japan (two life tables for 1899 and 1908, with respective $_{20}P_0$ at 0.70 and 0.69); (*b*) from the South pattern: (iv) Italy (five life tables for the period from 1881 to 1921, with $_{20}P_0$ ranging from 0.54 to 0.74, (v) Greece (1928 life table, with $_{20}P_0 = 0.74$), (vi) Portugal (three life tables for the period from 1920 to 1940, with $_{20}P_0$ ranging from 0.59 to 0.77), and (vii) Spain (1930 life table, with $_{20}P_0 = 0.75$); (*c*) from the Latin American pattern: (viii) El Salvador (1950 life table, with $_{20}P_0 = 0.77$), and (ix) Guatemala (1961 and 1969 life tables, with $_{20}P_0$ at 0.73 and 0.76, respectively); (*d*) from the Chilean pattern: (x) Chile (five life tables for the period from 1909 to 1950, with $_{20}P_0$ ranging from 0.55 to 0.76); (*e*) from the Far Eastern pattern: (xi) Taiwan, Province of China (three life tables for the period from 1920 to 1936, with $_{20}P_0$ ranging from 0.54 to 0.73); and (*f*) from sub-Saharan Africa, for which no single model mortality pattern had been established: (xii) South Africa (coloured population, 1941 and 1951 life tables, with respective $_{20}P_0$ at 0.68 and 0.73). Moreover, only for three model mortality patterns (South, Chilean and Far Eastern) are causes-of-death life tables available for the extreme points of this phase. Thus, the South Asian pattern is the only model mortality pattern for the developing countries for which appropriate data on causes of death are completely lacking. Fortunately, this model mortality pattern, which characterizes some of the most populous developing countries, is likely to have much in common with the European "South" pattern.

[11] The table below shows the composition of three large categories of causes of death:

Groups of causes of death from Preston, Keyfitz and Schoen (1972)

A.	Diseases of infancy and diarrhoeal diseases	(9)	Certain diseases of infancy
		(6)	Diarrhoea, gastritis, enteritis
B.	Respiratory diseases	(5)	Influenza, pneumonia, bronchitis
C.	Infectious and parasitic diseases	(2)	Other infectious and parasitic diseases
		(1)	Respiratory tuberculosis

Averaged across eight populations plotted on figure III, these three categories of diseases caused 79 per cent of all deaths in the first 20 years of life, as well as in each of the three underlying age intervals.

[12] It has to be noted that much higher levels of mortality from category C diseases in Guatemala in 1961 than in two other developing countries decades earlier may reflect differences in natural environments.

[13] The term "fertility goal" refers to the number of live (single) births (Bd) needed to assure parents that, given particular mortality conditions, no less than C_d of their children will survive until the relevant age (j), where C_d is the family size goal. By definition, B_d is not less than C_d and the difference between them depends on the mortality level (measured by the overall probability of survival from birth to age j), age schedule of mortality and the level of confidence in achieving the family size goal that is assumed to satisfy parents.

[14] This threshold level for the age of confidence is chosen because a usual inter-birth interval in the absence of contraception is about two years; the perception that a child's survival is virtually assured beyond this point is likely to initiate a straightforward wait-and-see strategy (i.e., replacement behaviour) in the form of deliberate spacing.

[15] Besides that, both fertility and mortality experience may be related to such common factors as age, education, socio-economic status and residence. Even with these controls, estimates may be biased upward because measured effects will include the fertility response to subsequent deaths, which are positively correlated with earlier deaths.

[16] A hazard model was used with all appropriate controls. The analysis was restricted only to women whose child, opening the interval, had survived.

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SEX DIFFERENTIALS IN SURVIVORSHIP IN THE DEVELOPING WORLD: LEVELS, REGIONAL PATTERNS AND DEMOGRAPHIC DETERMINANTS

United Nations Secretariat *

SUMMARY

The present study analyses patterns of sex differentials in survivorship in less developed countries (LDCs) and compares the findings to the historical (1850s to present) experience of the more developed world.

On average, the sex differential in life expectancy at birth (SDLE) increases by about one fifth of a year, in favour of females, for each one-year improvement in life expectancy at birth. At current average levels of life expectancy in the developing countries (59 years), the SDLE is estimated to be 3.2 years.

Even after controlling for differences in mortality level, strong regional variation exists in sex differentials. Within-continent variations in the Americas and Asia are mainly due to differences in female life expectancy. Within Africa, however, the variation in sex differentials is due to a combination of low female life expectancy and high male life expectancy. Within-continent variation is due to mortality differentials among all age groups. However, different age groups play greater or lesser roles within the different continents.

At high mortality levels, SDLEs, on average, are smaller in the developing countries than those historically found in the more developed countries (MDCs), but by the later stage of the mortality transition, the SDLEs in LDCs are, on average, similar to those found in the MDCs. The widening of the female-male differential in life expectancy at birth does not appear to be a concomitant consequence of mortality decline, and may, in fact, prove to be a post-Second World War phenomenon related to post-War changes in areas of personal behaviour, societal modernization and economic structure.

That women live longer than men is one of those few demographic facts known to lay persons and demographers alike. Only in a few countries, concentrated in South Asia and Northern Africa, do women, on average, die at earlier ages than men and in most of those countries this female disadvantage is minimal by the time of marriage and has disappeared by the end of the childbearing period.

The size of the female advantage in life expectancy varies among populations. However, this variation is not random but is systematically associated with geographical region and overall mortality levels (United Nations, 1983). Those associations pinpoint the relationship between sex differentials in survivorship and mortality patterns within societies and their interrelationship with the habits of culture and with modernization. Although likely of less importance, sex differentials also have some association with genetic traits of populations and the ecology of the land these populations inhabit. In summary, sex differentials are a geo-cultural phenomenon.

The sections that follow consider levels and regional patterns of sex differentials in life expectancy at birth, the role of each sex, age groups responsible for regional patterns, and patterns in the less developed world compared with those of the more developed countries.

THE DATA SETS

The data set used for the analysis of the less developed countries consists of 78 national life tables for the period 1945-1981 whose age-sex patterns of mortality have been evaluated as relatively reliable (United Nations, 1986a). The data set contains 13 life tables from Africa, 37 from the Americas and 28 from Asia. It represents a cross-section of mortality levels: 10 life tables are from populations that exhibit a life expectancy at birth (both sexes

* Population Division, Department of International Economic and Social Affairs.

combined) under 50 years, 10 from 50 to 55 years, 7 from 55 to 60 years, 23 from 60 to 65 years, 16 from 65 to 70 years, and 12 of 70 years or greater. All of these life tables are based on recorded age and sex patterns of mortality, as derived either from vital registration data combined with a census population, or from vital statistics and population data collected through sample surveys. In some cases, recorded mortality levels have been adjusted, based on the application of a variety of analytical techniques, because of incompleteness of the collected death data (relative to that of recorded births or population). The countries in the data set, with life expectancies at birth, by sex, are given in annex table 1. For purposes of analysis, the countries have been categorized by continent (Africa, America, Asia) and by United Nations region (Northern Africa and Other Africa on the African continent; Southern Asia, East Asia and Southeast Asia, and Western South Asia on the Asian continent; Temperate South America and Other America on the American continent). The life tables themselves and description of the sources of data and methods of construction are given in a previous publication (United Nations, 1986a).

The data set for the more developed world consists of 284 life tables from 28 countries, covering the period from the middle of the nineteenth century through the early 1980s. The life tables were compiled by the Population Division from two sources. Source 1 consists of the empirical life tables that were accepted by Coale and Demeny (1966) as the basis for construction of their model life table system; source 2 is based on the World Health Organization (WHO) data tape, which contains deaths and underlying population by age and sex. The source 1 life tables refer roughly to the period from the second half of the nineteenth century to the first half of the twentieth century. During this period, life expectancy increased from about 40 years at birth to about 70 years (Wrigley, 1976; United Nations, 1986a). Included in source 1 are life tables from Europe, the United States of America and Canada, Australia and New Zealand, and Japan. Source 1 also includes 10 life tables from populations that are generally considered developing, namely, Israel, South Africa and China (Province of Taiwan). These developing country life tables have been removed from the data set. Also omitted from the source 1 data set were life tables that referred to sub-national geographical areas, life tables from the same country whose reference dates overlapped or were very close and life tables from two small countries (Luxembourg and Iceland) which exhibited erratic age-sex patterns.

As the most recent life tables in the source 1 data set refer only to the late 1950s, this data set does not reflect the extensive widening of sex differentials in mortality that occurred in many developed countries during the 1960s. As a result, source 1 was updated with the WHO computerized file of registered deaths and underlying population by age and sex. This file (source 2) appended life tables for the more developed countries through the early 1980s.

The MDC data set consisted of 37 life tables that exhibited a life expectancy at birth under 50 years, 16 life tables between 50 and 55 years, 22 between 55 and 60 years, 23 between 60 and 65 years, 52 between 65 and 70 years, and 134 of 70 years or greater. Regional variation in mortality sex differentials is known to exist throughout the developed world (Lopez, 1983; Preston, 1976). However, as the focus of this paper is on less developed countries, the more developed countries are only considered in aggregate. Annex table 2 lists the life tables included, the life expectancy at birth and the source of data.

Sex differentials in survivorship

Survivorship in the present study is measured by life expectancy at birth, and general levels and trends of sex differentials are determined by regressions of the female-male differential in life expectancy on life expectancy at birth for both sexes combined.

The cross-sectional regression model for the 78 less developed countries is as follows:

$$SDLE_i = a + b(e[bs]_i - Z) + {}_i$$

Region	Slope (b)	Intercept around Z=59 (a−bZ)	Intercept around Z=74 (a−bZ)	R^2	(1)
Less developed countries	.203	3.23	6.27	.41	

(All coefficients are significant at the .0001 level)

where $SDLE_i$ is female life expectancy at birth less male life expectancy at birth for country i; (the sex differential in life expectancy), $e[bs]_i$ is the observed both sexes life expectancy at birth for country i, a and b are parameters (intercept and slope) to be estimated, Z is the point of reference for the intercept, and E_i is the statistical error term (residual) for the country.

The slope of the regression line indicates the expected increase in the female-male differential corresponding to a one-year increase in life expectancy at birth for both sexes combined. Two sets of intercepts are given: one based around a life expectancy at birth of 59 years, and one based around 74 years. The intercept then indicates the expected female-male differential (SDLE) when life expectancy at birth is at each of these levels. These two values were chosen to correspond approximately to current levels of survivorship in the less developed and more developed world, respectively.

The regression equation indicates that, on average, the SDLE increases by about one fifth of a year, in favour of females, for each one-year improvement in life expectancy at birth. At current average levels of life expectancy (59 years) in the developing countries, the SDLE is estimated to be 3.2 years; if future trends follow the current cross-sectional path, the female advantage will double to 6.3 years when overall life expectancy has reached 74 years. This latter life expectancy (equal to that currently found in the more developed world) is not projected to be reached by the less developed world as a whole until well after 2025.

The coefficient of determination indicates that the overall life expectancy explains about two fifths of the variation in the SDLE. That is, many of the countries in the data set exhibit differentials higher or lower than those predicted by the regression equation. Part of this variation is certainly due to data errors and random effects. However, the environmental, cultural and

behavioural patterns that were alluded to in the summary certainly play a role.

From the type of data available it is not possible to explain fully why some countries exhibit larger or smaller sex differentials than others. However, a first approach to identifying factors that might statistically explain variation in the SDLE is provided by analysis of the residuals ($_i$) from the regression equation (1); that is the difference between the actual and predicted sex differential for each country. Annex table 3 exhibits the regression residuals for each of the 78 life tables included, grouped according to United Nations region.

The residuals vary systematically by geographic location. The results of the classification are summarized in table 1 and in figure I. Even after controlling for differences in mortality level, strong regional variation exists in sex differentials. Females are most advantaged (or, correspondingly, males are most disadvantaged) in Temperate South America, Other Africa (defined here as all Africa except Northern Africa) and East/Southeast Asia. Females are most disadvantaged (or, correspondingly, males most advantaged) in Southern and Western South Asia and Northern Africa. Other America exhibits no distinctive pattern.

TABLE 1. REGIONAL VARIATION IN SEX DIFFERENTIALS IN LIFE EXPECTANCY AT BIRTH

Region	*Number of life tables in sample*	*Proportion of life tables with relatively high*	
		Female mortality	*Male mortality*
		(Negative residuals)	*(Positive residuals)*
AFRICA			
Northern Africa	5	0.80	0.20
Other Africa	8	0.12	0.88
ASIA			
Southern Asia	11	1.00	0
East Asia and Southeast Asia	12	0.25	0.75
Western South Asia	5	1.00	0
AMERICAS			
Temperate South America	7	0	1.00
Other America	30	0.47	0.53

Source: Annex table 3.

Although the above table succinctly summarizes the geographic variation, a boxplot (McNeil, 1977, pp. 6-7) permits a more intensive view of the situation. The boxplot in figure I presents, for each region, the interquartile range of the residuals (outliers are not indicated); the cross-bar indicates the median residual. The two regions with the largest negative residuals are Southern Asia and Western South Asia. Both portray similar patterns. The median residual (that is, the median amount by which the observed SDLE differs from that predicted) is −2.9 years for Southern Asia and −2.6 years for Western South Asia. The interquartile ranges are −3.5 to −2.4 and −3.0 to −1.6 respectively.

As indicated by table 1 and figure I, the Northern African countries also exhibit lower than average female-male differences but to a lesser extent than Southern and Western South Asia. The median divergence is −0.7 years and the interquartile range is from −1.7 to −0.4. It is important to note that the size of the SDLE in these regions is smaller than predicted in countries where mortality is low, such as Israel and Kuwait, as well as in countries where mortality remains high, such as Nepal and Bangladesh (see annex table 3).

At the other extreme are the African countries (except Northern Africa), East Asian and Southeast Asian countries and Temperate South American countries. It is the "Other Africa" region that exhibits the widest sex differentials in life expectancy at birth, a median of 3.0 years (greater than that predicted from the regression equation). The interquartile range for this region is, however, exceptionally large (0.8 to 4.5), reflecting not only the diversity within the African region but probably also the lower quality of African data in general. Survivorship differences are particularly large in Réunion and Cameroon (see annex table 3).

Sex differentials in life expectancy are also particularly wide in East and Southeast Asia, with a median excess differential of one year. What is most striking about the sex differentials in this region is a clear pattern over time in the size of the differentials (see annex table 3). Where a number of life tables are available for the same country, the excess differential declines over time. The earliest dated life tables exhibit among the widest female-male differentials. However, the more recent life tables exhibit no apparent differences from that expected on the basis of the regression. Life tables were available at two or more dates from Hong Kong, the Philippines and Singapore. For Hong Kong, excess female survivorship declines from 2.4 years to −0.6 years, for the Philippines from 1.6 years to 0.8 years and for Singapore from 2.0 years to −0.4 years (see annex table 3). This change is significant because the age-sex pattern of mortality in these countries had become known for their distinctive pattern of relatively high mortality at old ages and large sex differentials in favour of females. The uniqueness of this pattern had warranted their inclusion as a special model life table family in demographic research (United Nations, 1983); the passing of this pattern hence is of special note.

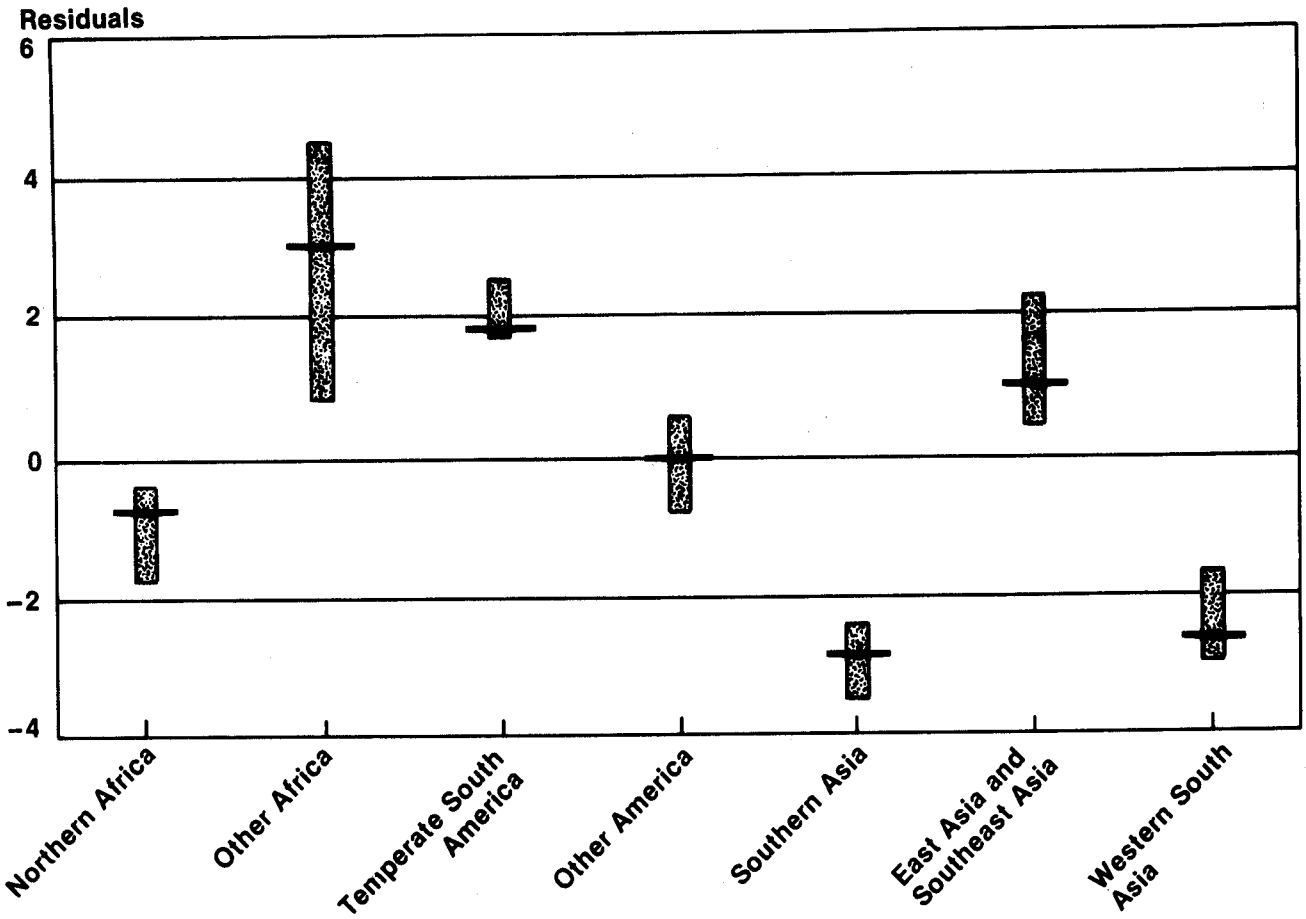
Temperate South America also exhibits particularly wide sex differentials in life expectancy in favour of females. The median sex differential (1.8 years higher than expected) is nearly as large as that in the Other African region, but perhaps more remarkable is its uniformity in size among the life tables. The interquartile range is 1.7 to 2.5 years.

The Other American countries present a pattern of sex differentials centred at the mean for all less developed countries. The median differential is only .02 years above that predicted from the regression.

WHICH SEX'S MORTALITY IS RESPONSIBLE FOR THE DIFFERENTIALS?

A female-male difference in life expectancy at birth can be particularly large (or small) because female life expectancy is greater (or less) than expected or male life expectancy is less (or greater) than expected, or some

Figure I. Interquartile range and median residuals for less developed regions



combination of the two. The difficulty in decomposing the female-male excess or deficit is that it is necessary to identify the "expected" male or female life expectancy.

Life expectancy at birth in the less developed world is well known to be related to levels of economic development (Preston, 1976 and 1986, for example) and, therefore, it would be reasonable to use an index of development as a predictor of male and female life expectancy for each country. We then become interested whether country (and regional) variation from expected life expectancy values varies by sex. If, for example, observed male life expectancy is as predicted based on the index of development but observed female life expectancy is higher than predicted, we would conclude that the mortality of females was responsible for the observed wide female-male difference in life expectancy. For the analysis, national per capita gross domestic product (GDP) (at constant 1975 prices) is chosen as the index of development. The GDP data refer to the same reference period as the life tables and are from United Nations sources (United Nations, 1984).

The approach adopted takes advantage of the contrasts found within each continent (see table 1). Among the countries of Africa, those in Northern Africa exhibited relatively high female mortality whereas the countries of Other Africa exhibited relatively high male mortality. The countries of Southern and Western South Asia exhibited relatively high female mortality whereas East Asia and Southeast Asia exhibited relatively high male mortality. Among the countries of the Americas, those of Temperate South America exhibited relatively high male mortality whereas those of Other America exhibited no distinctive sex pattern. Within each continent regression equations of the form

$$\hat{e}_0^s = a_s + b_s \ln Y + c_s \text{REGION}$$

were estimated where $\hat{e}^s$ is the predicted life expectancy at birth for sex s, Y is per capita income, REGION indicates the region within each continent and a_s, b_s and c_s are the sex-specific parameters to be estimated.[1]

The dummy variable REGION is defined for each continent as

REGION	*Africa*	*Americas*	*Asia*
0	Other Africa	Other Americas	East Asia and Southeast Asia
1	Northern Africa	Temperate South America	Southern and Western South Asia

The regression coefficients for the region variable (REGION) are exhibited in the following table:

	Regression coefficients		
Continent	*Female*	*Male*	*Difference of coefficients*
Africa			
Effect of Northern Africa on e_0 vs. Other Africa	−1.19	2.04	−3.23
Americas			
Effect of Temperate South America on e_0 vs. Other America	3.05	0.48	2.57
Asia			
Effect of Southern and Western Asia on e_0 vs. East Asia and Southeast Asia	−6.10	−0.95	−5.15

Although only one coefficient of the variable REGION is statistically significant (that for the Asian female sample, which is significant at the .01 level), the results remain of demographic interest. To the extent that the coefficients are representative of the universe from which they were drawn, Northern Africa exhibits a sex differential in life expectancy that is on average 3.2 years less than that in Other Africa, after controlling for income differences. This narrow sex differential appears to be due to a combination of effects—for a given level of per capita income, male life expectancy in Northern Africa is about two years greater than the rest of Africa but female life expectancy is about one and one fifth years less. In the other two continents, though, differences from predicted life expectancy for females provided the major reason for variation in sex differentials. Temperate South America's differential is about 2.6 years greater than the remaining American countries after controlling for income differences. No demographically significant difference in male life expectancy appears between Temperate South America and the remainder of the Americas; however, predicted female life expectancies are three years greater in Temperate South America. In other words, relative to other American countries, Temperate South America exhibits large sex differentials in life expectancy in favour of females, not because males exhibit particularly high mortality but because females are particularly advantaged. Differences from predicted life expectancy for females also plays the major role in Asia. The income-adjusted differential in life expectancy between females and males in Southern and Western South Asia is five years less than that of East and Southeast Asia. Females in Southern and Western South Asia exhibit a life expectancy at birth that is an astonishing six years lower than their East and Southeast Asian counterparts (after adjustment for level of GDP). Southern and Western South Asian males, on the other hand, have a life expectancy that is only one year lower.

In summary, regional variations in sex mortality differentials in the Americas and Asia appear to be due mainly to intracontinental differences in female life expectancy. In Africa, however, the relatively small sex differentials in Northern Africa appear to be due to a combination of low female life expectancies and high male life expectancies. In the next section these differentials are explored further by investigating the age groups responsible for the intracontinental regional differences.

Sex differentials in survivorship and age

Excess male or female mortality for a country or region may exhibit itself throughout the entire age span or be restricted to specific age groups. In the absence of reliable cause-of-death information for developing countries, age is a convenient proxy for the aetiology of the mortality differentials. This is because cause-of-death structure is closely associated with age. In "typical" developing countries, the causes of death prominent within each age group are as follows:

Age groups (in years)	*Dominant causes of death*
0-14	Respiratory diseases, infections and parasitic diseases
15-29	Accidents, suicides, violence, maternal causes
30-44	Accidents, suicides, violence, maternal causes
45-59	Cardio-vascular diseases, neoplasms
60-74	Cardio-vascular diseases, neoplasms
75+	Cardio-vascular diseases, neoplasms

Age is also a significant variable for study because of its role as a life-cycle marker. Differential survivorship during childhood, at ages of marital entry, during the childbearing years or during the late adult and elderly years are likely to raise different concerns and have different consequences.

The approach for studying age effects is decomposition by age of the intracontinental regional differences in the sex difference in life expectancy (SDLE). The process is two-staged. First, for each country, the SDLE is decomposed into that due to each of six 15-year age groups: 0-14, 15-29, 30-44, 45-59, 60-74 and 75+. The decomposition formula is identical to one used and described in previous United Nations studies (United Nations, 1982 and 1983) and is described therein. For each country, the SDLE is equal to the sum of the six age components:

$$\text{SDLE} = \sum_{x} {}_{15}\Delta_x,\ x = 0, 15, \ldots, 75$$

where ${}_{15}\Delta_x$ is the amount of the SDLE accounted for by age group $(x,x+15)$. For the second stage, a pair of regressions is calculated for each of the three continents. Each ${}_{15}\Delta_x$ value is regressed on life expectancy at birth for both sexes combined and the dummy variable for the region (REGION) used previously. A parallel regression

is calculated with SDLE as the dependent variable. The regressions are therefore

$$\hat{}_{15}\Delta_x = a_x + b_x e_{bs} + c_x \text{REGION}$$

$$\hat{\text{SDLE}} = A + Be_{bs} + C\ \text{REGION}$$

The coefficient C indicates the average effect (additive) on the SDLE of being a Northern African country (for the Africa regression), Temperate South American country (for the Americas regression) or Southern and Western South Asian country (for the Asia regression). The coefficient c_x indicates the same effect for each age component of the sex differential, ${}_{15}\Delta_x$. Because of the linearity of the regression model, $\sum_x c_x = C$. That is, the total intracontinental regional effect (C) can be decomposed into that due to each of the six age groups. This decomposition of C into its age components indicates whether the intracontinental regional differences are primarily due to certain age groups or whether all age groups play roughly equal roles. The results of this analysis are summarized in the following table and in figure II.

It is clear from this table that regional variations within each continent are due to mortality differentials among all age groups. However, different age groups play greater or lesser roles within the different continents. Nearly two fifths of the intracontinental variation in SDLE among African countries is due to age group 0-14. The variations in the Americas are due mainly to ages 45-74. About two thirds of the regional differential in the Americas is due to these age groups. Unlike Africa and the Americas, regional variation of the SDLE in Asia is due both to the variation in childhood ages (0-14) and the late adult ages of 45-74. The difference in the latter group accounts for 50 per cent of the relative excess female mortality.

Since the pattern of cause of death varies according to age, sex differentials are probably due to a wide variety of causes that vary from region to region. Regional differences in the level of sex differences in Africa are due slightly more to differences in the early ages where the childhood respiratory diseases, infections and parasitic diseases generally dominate. In the Americas, differences in the late adult years play a strong role in explaining regional differences implying a significant role for cardio-vascular diseases and neoplasms. The Asian countries exhibit no distinctive age pattern, perhaps indicating that sex differences are strong through the entire cause-of-death structure.

Developing country sex differentials in the historical developed country context

Sex differentials exist in less developed countries (LDCs) and more developed countries (MDCs) alike. The United Nations (1986b) projects for the period 1985-1990 a female survivorship advantage of 7.4 years in the more developed world and a two-year advantage in LDCs.

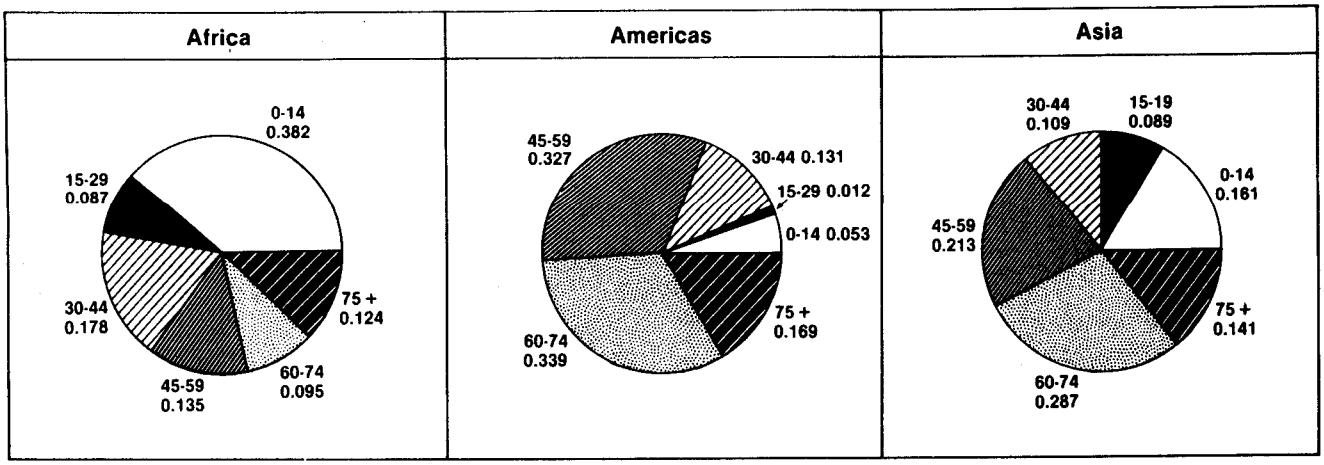
Hence, differentials are currently much wider in more developed countries. But life expectancy at birth, in general, is higher in MDCs than in LDCs. Since sex differentials widen in favour of females during the mortality transition, the MDC-LDC disparity may be due to the stage of the mortality transition. The present section explores LDC sex differentials within the context of the historical MDC experience.

To study this issue the MDC data set described earlier will be considered in two subsets: life tables referring to

Extent of variation in female-male difference in life expectancy (SDLE) due to age group

Continent	Total (C)	0-14 (c_0)	15-29 (c_{15})	30-44 (c_{30})	45-59 (c_{45})	60-74 (c_{60})	75+ (c_{75})
	Absolute numbers in years						
Africa							
Northern Africa compared to Other Africa	−3.603	−1.375	−.312	−.641	−.485	−.343	−.446
America							
Temperate South America compared to Other America	2.168	.114	.025	.283	.709	.734	.303
Asia							
Southern and Western South Asia compared to East and Southeast Asia	−3.927	−.633	−.348	−.427	−.836	−1.128	−.555
	Proportional distribution						
Africa							
Northern Africa compared to to Other Africa	1.000	.382	.087	.178	.135	.095	.124
America							
Temperate South America compared to Other America	1.000	.053	.012	.131	.327	.339	.169
Asia							
Southern and Western South Asia compared to East and Southeast Asia	1.000	.161	.089	.109	.213	.287	.141

Figure II. Role of age groups in explaining intracontinental variation in sex differentials



before 1945 and life tables referring to the period from 1945 to the present. The first subset includes MDC life tables in which life expectancies range from under 30 years to about 65 years. These life tables therefore encompass a range of mortality levels that overlap those found in the LDC data set. These life tables, however, refer to a time period (from the middle of the nineteenth century to the end of the Second World War) that precedes the development of life-styles and the environmental changes related to the urban-industrial-modern milieu and the clinic-based health revolution that affected MDCs and LDCs alike after the Second World War. The second subset includes MDC life tables in which life expectancies vary roughly from 65 years to over 75, life expectancies higher in general than those in the LDC sample. This subset, however, refers to a time period identical to that available for the LDCs.

The cross-sectional regression of the SDLE on life expectancy at birth is as follows:

$$SDLE_i = a + b(e[bs]_i - Z) + i$$

Data set	*Slope (b)*	*Intercept around* $Z=45$ $(a - bZ)$	$Z=59$ $(a - bZ)$	$Z=74$ $(a - bZ)$	R^2	
LDCs	.203	0.39	3.23	6.27	.4090	
MDCs, pre-1945	.003	2.84	2.88	2.92	.0005	(2)
MDCs, 1945-present.	.107	2.90	4.40	6.02	.0772	

(All slope coefficients are significant at the p = .0001 level except for that of the pre-1945 MDC data set (p = .83).)

Life expectancy at birth explains none of the variation in the sex differential among the pre-1945 more developed country life tables. The coefficient of determination and the slope are both not statistically different from zero. Up until the Second World War, the female-male difference in life expectancy at birth was relatively steady at about three years. Life expectancy explained only a little more of the differential after 1945; roughly 8 per cent of the variation in the SDLE is due to life expectancy and an improvement of one year in life expectancy is associated with just one tenth of a year increase in the sex differential.

The sensitivity of the sex differential to overall survivorship level is much higher in the case of the LDCs, where the coefficient of determination reached 0.4 and the slope is 0.2. The LDC regression equation indicates that the sex differential widened, on average, from about 0.4 years when life expectancy was 45 years at birth to about 3.2 years when life expectancy is at 59 years. At low levels of life expectancy, therefore, LDC sex differentials were narrow (0.4 years) in comparison to historical MDC experience (which averaged about 2.8-2.9 years when life expectancy was 45 years). At the higher level of life expectancy (59 years, similar to the average levels of LDCs today) the predicted female-male difference in LDCs falls between that predicted by the two MDC equations. The LDC equation extrapolated to current MDC levels of life expectancy yields a female-male difference of 6.3 years, not far from that predicted by the post-1945 MDC regression.

In summary, these regressions indicate that at high mortality levels, LDC sex differentials, on average, are smaller than those historically found in the MDCs, but by the later stage of the mortality transition, the LDC sex differentials are, on average, similar to those found in the MDCs.

The MDC equations indicate that time period may play a role in explaining the size of sex differentials. Time period, of course, would here be a proxy for other characteristics of society such as urbanization, life-styles or per capita income. To assess the significance of time, additional regressions were prepared which included "decade" variables along with life expectancy. Regressions were carried out on the LDC data set, the total MDC data set and the two MDC subsets. The results of these regressions follow:

REGRESSIONS OF SEX DIFFERENTIAL IN LIFE EXPECTANCY ON LIFE EXPECTANCY AND TIME PERIOD

$$SDLE_i = a + b(e[bs]_i) + C_jDEC_j$$

Variable	Coefficients				
	LDC	Total MDC	Pre-1945 MDC	Post-1945 MDC	
e[bs] (life expectancy)	.205[a]	−.007	.006	−.061[a]	
DEC_1 (before 1900)...	x	−3.157[a]	0	x	
DEC_2 (1900-1944).....	x	−3.175[a]	−.142	x	
DEC_3 (1945-1954).....	−1.778	−1.627[a]	x	−1.880[a]	(3)
DEC_4 (1955-1964).....	.103	−.707[a]	x	−.777[a]	
DEC_5 (1965-1974).....	0	0	x	0	
DEC_6 (1975+).....	−.780	.601[a]	x	.697[a]	
R^2	.441	.654	.003	.351	

NOTE: In all regressions, the DEC_j variables are dummy variables. For the LDC, total MDC and post-1945 MDC runs, DEC_5 is the omitted variable. For the pre-1945 MDC sample, DEC_1 is the omitted variable.

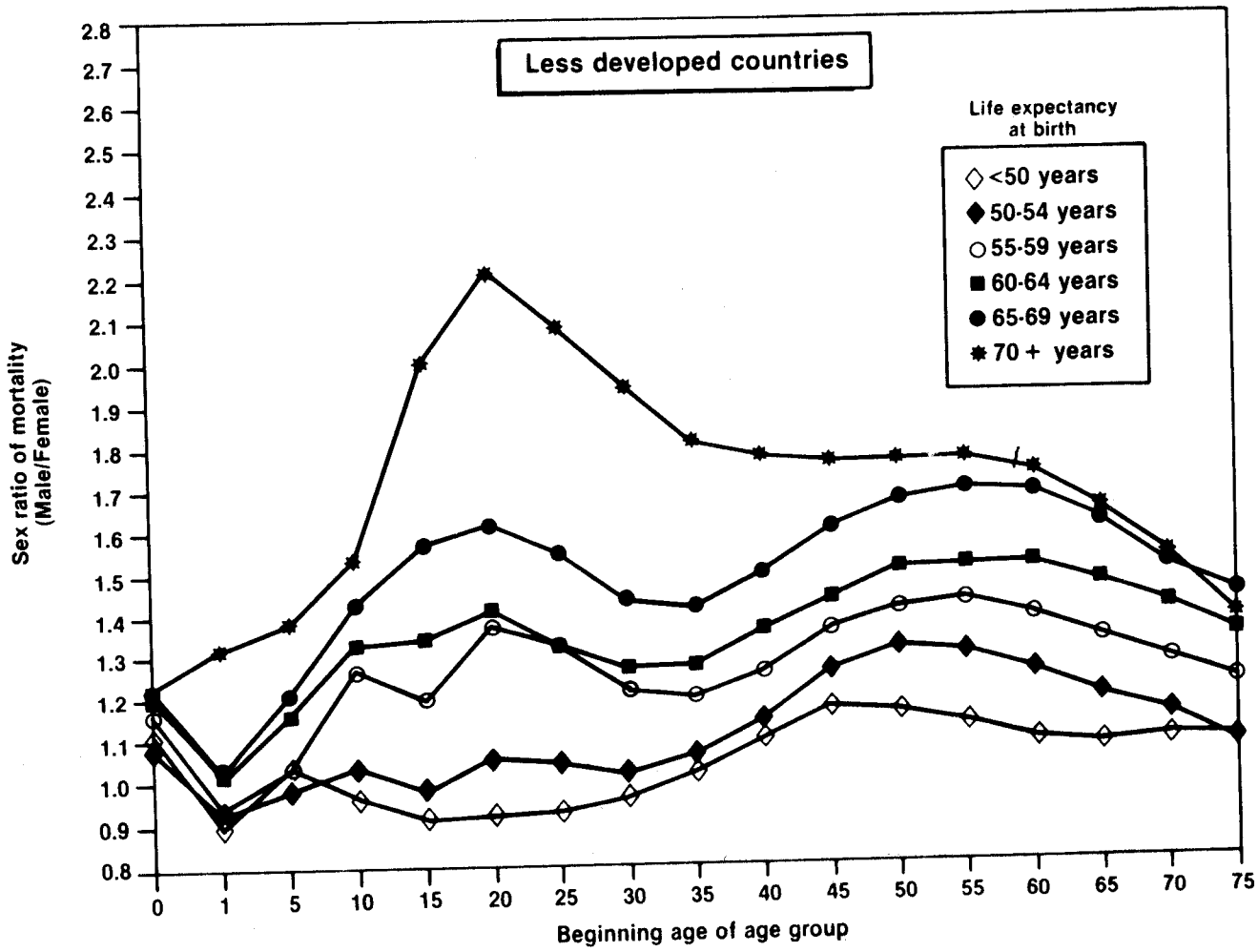
[a] Significant at the .05 level.

Comparisons of these regressions with those presented previously indicate that time period plays no statistically significant role in explaining sex differentials among the less developed countries or among the pre-1945 MDCs. However, among the total MDC sample and the post-1945 MDC sample, time has both a demographic and statistically significant effect, with differentials becoming wider over time. The coefficients of life expectancy in equation set (3) are further reduced for the MDC samples compared to the previous ones (equation set (2) above).

It appears, therefore, that among LDCs sex differentials in life expectancy are sensitive to changes in overall mortality level.[2] In MDCs, however, the widening of sex differentials is not related to the level of life expectancy but instead is related to factors exogenous to the mortality transition as proxied by time period.

These results can be extended by examining the relationship of age-specific sex ratios in mortality with life

Figure III. Age-specific sex ratios

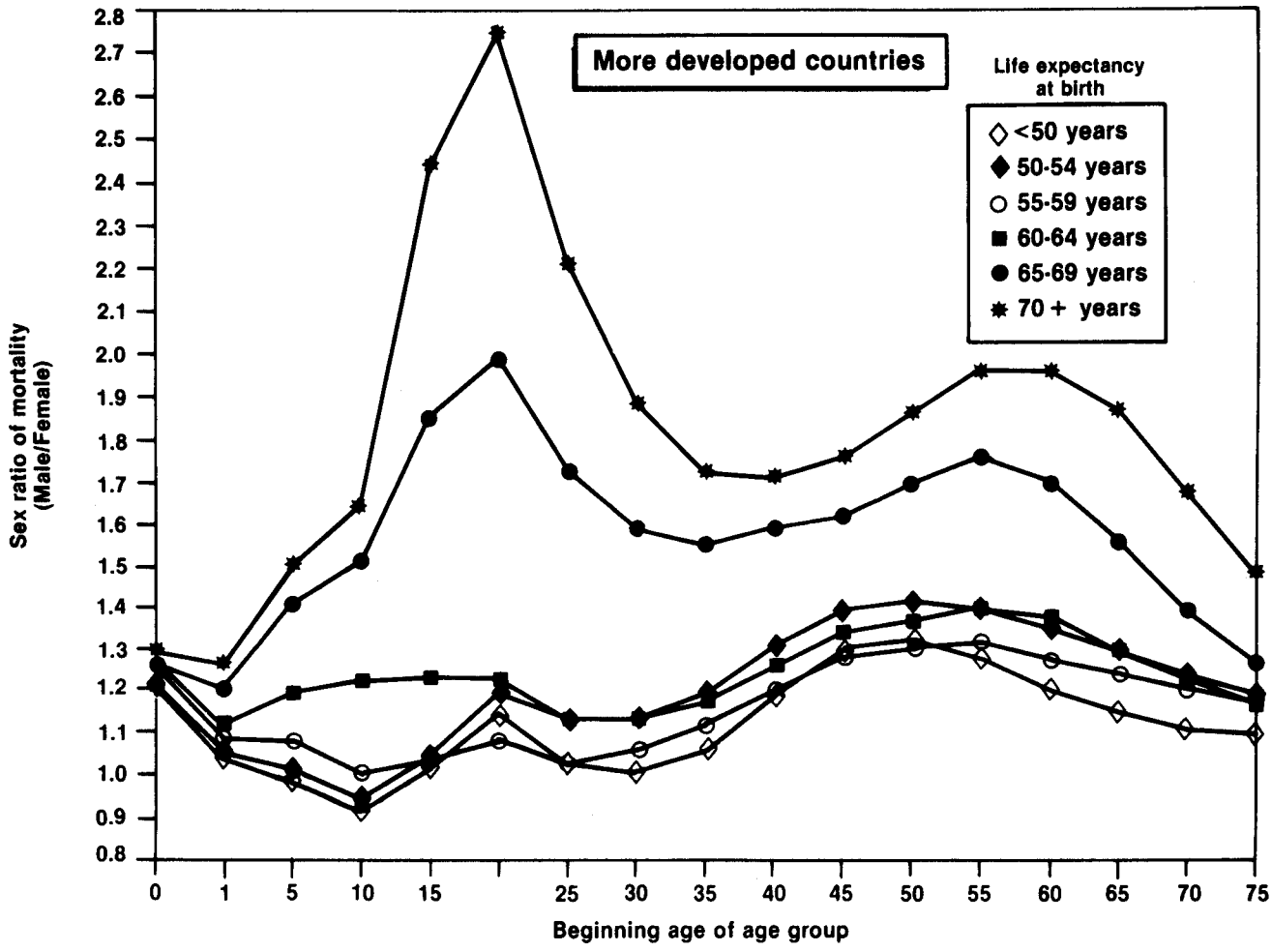


expectancy for LDCs and MDCs. Figures III and IV present graphically the average patterns of age-specific sex ratios (males to females) of central death rates in five groupings of life expectancy at birth for the LDC and MDC data sets. For both data sets, the age curve of sex ratios changes from an elongated U-shaped curve to an inverted U-shaped curve during the period of the mortality transition. The change in shape is due to the large rise in sex ratios for the early childbearing ages. Sex ratios double for ages 15-34 in the LDCs and for ages 15-29 in the MDCs, as life expectancy moves from less than 50 years at birth to over 70 years.

The rise is undoubtedly due to a yet unmeasured mix of improving maternal mortality for females and constant (or even rising) mortality from accidents and violence for males. Besides the emerging hump centred on the early childbearing years, there is also a secondary hump that appears in both the LDC and MDC patterns, falling approximately between ages 50 and 65. The secondary hump is small in the LDCs but quite pronounced among the MDC populations.

Comparison of the age curves for the "e_0 under 50" group and "e_0 greater than 70" group indicates the overall pattern of age changes in sex ratios. For the LDCs, changes are small under age 10, compared to older ages. Sex ratios increase by at least 0.5 for nearly every age group older than 10 years, with, as indicated above, the maximum change being during the early childbearing ages. Sex ratio changes for the MDCs have been much less striking: increases of 0.5 or greater appear only for ages 5 through 44. Changes in sex ratios for the younger and older ages are substantially smaller. From further examination of figures III and IV it can be seen that for the LDC populations sex ratios rise consistently and by more or less equal amounts as life expectancy at birth progresses from one level to the next.

Figure IV. Age-specific sex ratios



For the MDCs, a different pattern emerges. Sex ratios in mortality remain nearly constant as survivorship improves for the "e_0 less than 50 years" group to the "e_0 between 60 and 65 years" group. Age-specific sex ratios rose for the more developed world only after average life expectancy reached 65 years at birth (which occurred during the first few years after the Second World War). This also leads to a change in the relationship between LDC and MDC sex ratios. At ages under 10, MDC sex ratios are larger than those in LDCs for all levels of survivorship. But for older ages the pattern is very different: MDC sex ratios are larger for lower survivorship levels but smaller for higher levels of survivorship. The cross-over generally occurs at the "e_0 between 55 and 60 years" group and is due to the aforementioned lack of rise in sex ratios among the MDC populations until after life expectancy at birth exceeds 65 years.

The results of this section indicate that changes in sex ratios of mortality are not necessarily a concomitant consequence of mortality decline, and may, in fact, be more closely related to "time period". Large rises in sex ratios may prove to be a post-Second World War phenomenon which, in general, has affected LDCs and MDCs alike. Post-War changes in areas of personal behaviour (smoking, exercise, violence), societal modernization (rapid urbanization, especially into large cities; motor vehicles), and economic structure (industrial accidents) are likely to be important explanatory variables.

Conclusion

In developing countries, the sex differential in life expectancy has widened in favour of females during the mortality transition. A regression based on a cross-national analysis of life tables from less developed countries for the period 1945 to 1981 indicates that a one-year gain in life expectancy at birth in LDCs corresponds approximately to a one-fifth-year gain in the sex differential. Sex differentials appeared to be narrowest (least favourable to females) in Southern and Western South Asia and Northern Africa; sex differentials were widest in East and Southeast Asia, Temperate South America and the African countries, excluding Northern Africa. Sex differentials in life expectancy in the more developed countries have also widened over time, at least during the post-Second World War period. In contrast to the LDCs, MDC sex differentials appear to be unrelated to overall level of life expectancy but to be related instead to factors exogenous to the mortality transition as proxied by a time variable.

A comparison of the MDC and LDC data sets indicates that at high mortality levels, less developed countries exhibited smaller differentials than those historically found in the more developed world. However, at moderate and low levels of mortality, sex differentials may not differ much between developed and developing countries. This general result, of course, must be considered within the context of significant geographic variation of sex differentials within both more developed and less developed regions.

As the overall sex differential in life expectancy widens in favour of females, the age pattern of the sex ratios of mortality (male central death rates divided by female rates), changes from a broad U-shaped curve to an inverted U-shaped curve. Among developing countries, this is due to the sharp rises in the sex ratio of mortality for age groups 15-34; in the developed countries, to a sharp rise among the ages 15-24. In LDCs, age-specific sex ratios in mortality have risen consistently as life expectancy at birth has risen from less than 50 years to over 70 years. Among MDCs, only insignificant rises in sex ratios occurred until average survivorship levels exceeded about 65 years at birth. For life tables with high mortality levels, age-specific sex ratios of mortality are greater in the more developed countries; for countries with lower mortality levels, the sex ratios of mortality are greater in the less developed countries.

The widening of sex differentials in survivorship and mortality is probably a post-Second World War phenomenon, in MDCs and LDCs alike, and may be related to a life-style and societal changes rather than being a "natural concomitant" of a mortality transition.

Notes

[1] Sri Lanka, Cuba, Guadeloupe, Martinique, Puerto Rico and Trinidad and Tobago were omitted from the regressions because of lack of national income data. Kuwait was omitted because its national income figures are greatly affected by asset sales (petroleum).

[2] Sex differentials in life expectancy remain statistically related to overall life expectancy among the LDCs even after control for regional variation. A regression of the sex differential on life expectancy at birth and five dummy variables representing regions (Other America is the omitted variable) found a coefficient of life expectancy .161 ($p < .0001$). The coefficient of determination (R^2) for the regression was .81.

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ANNEX

TABLE 1. DEVELOPING COUNTRY LIFE TABLES AND LIFE EXPECTANCY AT BIRTH, BY SEX

Country and region	Reference period	Life expectancy at birth: Males	Life expectancy at birth: Females
Africa			
Northern Africa			
Algeria	1969-1971	52.89	53.18
Egypt	1965-1967	50.26	51.48
Egypt	1975-1977	53.15	55.90
Morocco	1972	51.70	52.85
Tunisia	1968-1969	52.71	52.45
Other Africa			
Burundi	1970-1971	43.23	44.15
Cameroon	1958-1965	36.01	39.66
Liberia	1970-1971	46.63	49.98
Mauritius	1961-1963	58.75	62.17
Mauritius	1971-1973	60.73	65.65
Réunion	1961-1962	53.61	60.71
Réunion	1967-1968	57.52	64.90
Réunion	1970	57.54	66.08
Americas			
Temperate South America			
Argentina	1959-1961	62.02	68.20
Argentina	1969-1970	62.08	69.65
Argentina	1979	65.97	73.12
Chile	1959-1961	54.75	60.14
Chile	1969-1971	58.87	64.93
Uruguay	1962-1964	65.76	71.84
Uruguay	1974-1976	65.78	72.75
Other America			
Colombia	1963-1965	56.98	59.69
Costa Rica	1962-1964	60.83	63.80
Costa Rica	1972-1974	67.48	71.05
Cuba	1969-1971	70.28	73.68
El Salvador	1970-1972	54.79	59.87
Guadeloupe	1966-1968	62.14	67.43
Guadeloupe	1981-1983	68.88	76.02
Guatemala	1963-1965	46.85	48.01
Guatemala	1972-1973	53.83	55.63
Guyana	1959-1961	59.51	63.34
Guyana	1969-1971	61.98	66.66
Honduras	1960-1962	40.61	44.12
Honduras	1973-1975	50.18	54.34
Jamaica	1959-1961	62.12	66.43
Jamaica	1969-1971	65.84	70.07
Martinique	1960-1962	62.53	66.51
Martinique	1966-1968	64.40	69.69
Martinique	1981-1983	70.98	75.36
Mexico	1969-1971	58.78	62.93
Panama	1960-1970	64.46	67.69
Panama	1970-1980	67.59	69.84
Puerto Rico	1949-1951	58.81	61.47
Puerto Rico	1959-1961	66.79	71.45
Puerto Rico	1969-1971	68.97	75.28
Puerto Rico	1979-1981	70.24	77.58
Suriname	1963-1964	62.08	66.39
Trinidad and Tobago	1945-1947	52.96	55.77
Trinidad and Tobago	1959-1961	62.34	66.57
Trinidad and Tobago	1969-1971	63.59	67.84
Venezuela	1970-1972	62.26	67.55
Asia			
Southern Asia			
Bangladesh	1964-1965	49.63	47.21
Bangladesh	1981	51.36	50.10
India	1971-1976	48.79	47.38
Iran	1973-1976	57.19	56.56
Nepal	1974-1976	44.85	42.07
Pakistan	1962-1965	49.75	48.55
Pakistan	1968-1971	52.89	50.24
Sri Lanka	1945-1947	44.82	43.08
Sri Lanka	1952-1954	58.37	57.26
Sri Lanka	1962-1964	62.14	62.63
Sri Lanka	1970-1972	63.82	66.72
East Asia and Southeast Asia			
Hong Kong	1960-1962	63.73	71.08
Hong Kong	1970-1972	66.84	75.15
Hong Kong	1976	69.62	76.53
Hong Kong	1981	72.03	77.92
Malaysia (Peninsular)	1969-1971	61.67	65.86
Philippines	1969-1971	58.68	64.00
Philippines	1974-1976	58.27	62.63
Republic of Korea	1971-1975	59.27	66.11
Singapore	1956-1958	60.51	66.68
Singapore	1969-1971	66.02	72.15
Singapore	1979-1981	68.84	74.26
Thailand	1969-1971	56.51	60.79
Western South Asia			
Israel	1960-1962	70.60	72.75
Israel	1971-1973	70.37	73.59
Kuwait	1974-1976	65.92	70.32
Kuwait	1979-1981	69.06	73.09
Syrian Arab Republic	1976-1978	61.53	62.35

Source: Age Structure of Mortality in Developing Countries: A Data Base for Cross-sectional and Time Series Research (ST/ESA/SER.R/66).

TABLE 2. DEVELOPED COUNTRY LIFE TABLES, LIFE EXPECTANCY AT BIRTH BY SEX, AND SOURCE OF DATA

Country	Reference period	Life expectancy at birth: Males	Life expectancy at birth: Females	Source of data[a]
Australia	1881-1890	47.23	50.92	C&D
	1890-1900	51.12	54.77	C&D
	1901-1910	55.23	58.86	C&D
	1920-1922	59.22	63.37	C&D
	1932-1934	63.59	67.24	C&D
	1946-1948	66.17	70.74	C&D
	1953-1955	67.24	72.85	C&D
	1960-1964	67.85	74.18	L&T
	1965-1969	67.71	74.35	L&T
	1970-1974	67.99	74.81	L&T
	1975-1979	69.98	77.03	WHO
	1980-1982	71.28	78.41	WHO
Austria	1900-1904	37.75	39.88	C&D
	1906-1910	40.71	42.88	C&D
	1930-1933	54.53	58.59	C&D
	1949-1951	61.97	67.00	C&D
	1960-1964	66.27	72.63	L&T
	1965-1969	66.59	73.28	L&T
	1970-1974	66.91	74.02	L&T
	1975-1979	68.27	75.34	WHO
	1980-1983	69.24	76.40	WHO
Belgium	1880-1890	43.68	46.66	C&D
	1891-1900	45.42	48.93	C&D
	1928-1932	56.10	59.82	C&D
	1946-1949	62.09	67.35	C&D
	1960-1964	67.31	73.30	L&T
	1965-1969	67.62	73.85	L&T
	1970-1974	68.21	74.67	L&T
	1975-1979	69.32	75.84	WHO
	1980-1981	69.95	76.74	WHO
Bulgaria	1960-1964	68.30	71.85	L&T
	1965-1969	68.86	72.89	L&T
	1970-1974	68.84	73.31	L&T
	1975-1979	68.59	73.56	WHO

TABLE 2 (*cont.*)

Country	Reference period	Life expectancy at birth		Source of data[a]
		Males	Females	
Bulgaria (*cont.*)	1980-1982	68.57	74.03	WHO
Canada	1926-1930	57.76	59.80	C&D
	1930-1932	60.05	62.13	C&D
	1940-1942	63.01	66.34	C&D
	1945	64.79	68.16	C&D
	1950-1952	66.44	70.93	C&D
	1956	67.78	72.95	C&D
	1960-1964	68.42	74.36	L&T
	1965-1969	68.90	75.42	L&T
	1970-1974	69.44	76.60	L&T
	1975-1979	70.51	77.95	WHO
	1980-1982	71.80	78.98	WHO
Czechoslovakia	1899-1902	38.87	41.72	C&D
	1929-1932	51.95	55.21	C&D
	1956-1958	67.12	72.05	C&D[b]
	1960-1964	67.62	73.29	L&T
	1965-1969	66.99	73.46	L&T
	1970-1974	66.64	73.56	L&T
	1975-1979	67.05	74.21	WHO
	1980-1982	67.00	74.35	WHO
Denmark	1895-1900	50.25	53.26	C&D
	1901-1905	53.02	56.29	C&D
	1906-1910	55.03	58.02	C&D
	1911-1915	56.28	59.27	C&D
	1921-1925	60.36	61.99	C&D
	1926-1930	61.02	62.66	C&D
	1931-1935	62.07	63.86	C&D
	1936-1940	63.61	65.87	C&D
	1941-1945	65.74	67.82	C&D
	1946-1950	67.92	70.28	C&D
	1951-1955	69.93	72.65	C&D
	1960-1964	70.46	74.46	L&T
	1965-1969	70.54	75.23	L&T
	1970-1974	70.98	76.40	L&T
	1975-1979	71.53	77.42	WHO
	1980-1983	71.48	77.50	WHO
England and Wales	1871-1880	41.35	44.64	C&D
	1891-1900	44.19	47.82	C&D
	1901-1910	48.58	52.45	C&D
	1910-1912	51.54	55.38	C&D
	1920-1922	55.65	59.61	C&D
	1930-1932	58.77	62.89	C&D
	1948-1951	66.19	71.08	C&D[c]
	1957-1959	68.11	73.87	C&D
	1960-1964	68.16	74.12	L&T
	1965-1969	68.66	74.91	L&T
	1970-1974	69.13	75.37	L&T
United Kingdom: England and Wales	1975-1979	69.99	76.08	WHO
	1980-1983	71.18	77.16	WHO
Finland	1881-1890	41.41	44.19	C&D
	1946-1950	58.67	65.94	C&D
	1951-1955	63.46	69.89	C&D
	1960-1964	65.51	72.51	L&T
	1965-1969	65.76	73.35	L&T
	1970-1974	66.56	75.00	L&T
	1975-1979	68.08	76.95	WHO
	1980-1983	69.83	78.23	WHO
France	1875-1877	41.89	44.03	C&D
	1880-1882	41.29	43.73	C&D
	1885-1887	41.76	44.44	C&D
	1898-1903	45.32	48.70	C&D
	1908-1913	48.53	52.42	C&D
	1920-1923	52.19	56.06	C&D
	1928-1933	54.33	59.07	C&D
	1933-1938	55.97	61.70	C&D
	1946-1948	62.58	68.01	C&D
	1950-1951	63.67	69.42	C&D
France (*cont.*)	1952-1956	65.11	71.24	C&D
	1958-1959	67.09	73.56	C&D[d]
	1960-1964	67.79	74.63	L&T
	1965-1969	68.18	75.65	L&T
	1970-1974	69.26	76.96	L&T
	1975-1979	70.10	78.13	WHO
	1980-1982	70.97	79.10	WHO
Germany	1871-1880	35.62	38.52	C&D
	1881-1890	37.22	40.28	C&D
	1891-1900	40.61	44.01	C&D
	1901-1910	44.88	48.37	C&D
	1910-1911	47.46	50.73	C&D
	1924-1926	56.03	58.84	C&D
	1932-1934	59.93	62.88	C&D
	1949-1951	64.63	68.55	C&D
	1955-1957	66.31	70.86	C&D[e]
German Democratic Republic	1960-1964	67.31	72.20	L&T
	1965-1969	68.11	73.19	L&T
	1970-1974	68.43	73.91	L&T
	1975-1979	68.82	74.62	WHO
	1980-1983	69.06	75.00	WHO
Germany, Federal Republic of	1960-1964	67.10	72.73	L&T
	1965-1969	67.47	73.51	L&T
	1970-1974	67.66	74.10	L&T
	1975-1979	68.94	75.58	WHO
	1980-1983	70.32	77.00	WHO
Greece	1960-1964	70.14	73.73	L&T
	1965-1969	70.93	74.81	L&T
	1970-1974	71.95	76.08	L&T
	1975-1979	72.57	76.98	WHO
	1980-1982	73.24	77.84	WHO
Hungary	1960-1964	66.40	70.91	L&T
	1965-1969	66.94	72.02	L&T
	1970-1974	66.52	72.40	L&T
	1975-1979	66.35	72.77	WHO
	1980-1983	65.42	72.99	WHO
Ireland	1925-1927	57.41	57.98	C&D
	1935-1937	58.35	59.82	C&D
	1940-1942	58.98	60.94	C&D
	1945-1947	60.44	62.32	C&D
	1950-1952	64.55	67.12	C&D
	1960-1964	68.17	71.97	L&T
	1965-1969	68.59	72.91	L&T
	1970-1974	68.71	73.59	L&T
	1975-1979	69.31	74.53	WHO
	1980-1981	70.00	75.47	WHO
Northern Ireland	1925-1927	55.50	56.19	C&D
	1957-1959	67.67	72.26	C&D
	1960-1964	67.77	72.65	L&T
	1965-1969	68.11	73.53	L&T
	1970-1974	67.28	73.77	L&T
United Kingdom: Northern Ireland	1975-1979	67.94	74.33	WHO
	1980-1982	69.30	75.56	WHO
Italy	1881-1882	35.16	35.66	C&D
	1899-1902	42.89	43.23	C&D
	1901-1911	44.27	44.87	C&D
	1910-1912	46.59	47.37	C&D
	1960-1964	67.04	72.34	L&T
	1965-1969	67.85	73.59	L&T
	1970-1974	69.02	75.07	L&T
	1975-1979	70.34	76.68	WHO
	1980	70.96	77.46	WHO
Japan	1950-1952	59.62	63.09	C&D
	1960-1964	66.67	71.62	L&T

TABLE 2 (*cont.*)

Country	Reference period	Life expectancy at birth		Source of data[a]
		Males	Females	
Japan (*cont.*)	1965-1969	68.78	73.99	L&T
	1970-1974	70.55	75.87	L&T
	1975-1979	72.74	77.96	WHO
	1980-1983	74.06	79.58	WHO
Netherlands	1870-1879	38.43	40.71	C&D
	1880-1889	42.47	45.05	C&D
	1890-1899	46.25	49.02	C&D
	1900-1909	51.01	53.47	C&D
	1921-1930	62.07	63.49	C&D
	1931-1940	65.78	67.23	C&D
	1947-1949	69.49	71.55	C&D
	1951-1955	71.04	73.59	C&D
	1960-1964	71.33	75.84	L&T
	1965-1969	71.11	76.39	L&T
	1970-1974	71.19	77.00	L&T
	1975-1979	71.96	78.40	WHO
	1980-1983	72.78	79.45	WHO
New Zealand (White)	1880-1892	54.50	57.37	C&D
	1891-1895	55.29	58.11	C&D
	1896-1900	57.36	59.96	C&D
	1901-1905	58.10	60.54	C&D
	1906-1910	59.17	61.76	C&D
	1911-1915	60.97	63.39	C&D
	1921-1922	62.98	65.64	C&D
	1925-1927	64.14	66.60	C&D
	1934-1938	65.56	68.53	C&D
	1950-1952	68.42	72.55	C&D
	1955-1957	69.00	74.03	C&D
New Zealand	1960-1964	68.52	73.99	L&T
	1965-1969	68.28	74.40	L&T
	1970-1974	68.70	74.91	L&T
	1975-1979	69.40	76.12	WHO
	1980-1983	70.53	76.58	WHO
Norway	1856-1865	47.48	50.06	C&D
	1871-1881	48.38	51.32	C&D
	1946-1950	69.30	72.78	C&D
	1951-1955	71.29	74.85	C&D
	1960-1964	71.15	75.92	L&T
	1965-1969	71.22	76.72	L&T
	1970-1974	71.36	77.64	L&T
	1975-1979	72.15	78.52	WHO
	1980-1983	72.65	79.44	WHO
Poland	1931-1932	48.19	51.46	C&D
	1948	55.67	61.53	C&D
	1952-1953	58.63	63.99	C&D
	1960-1964	65.03	71.01	L&T
	1965-1969	66.68	73.01	L&T
	1970-1974	67.04	74.01	L&T
	1975-1979	66.83	74.68	WHO
	1980-1983	66.88	75.04	WHO
Portugal	1919-1922	35.84	40.07	C&D
	1929-1932	44.86	49.31	C&D
	1939-1942	48.61	52.85	C&D
	1944-1947	51.30	56.38	C&D
	1949-1952	55.58	60.68	C&D
	1955-1958	59.21	64.35	C&D[f]
	1960-1964	61.96	67.51	L&T
	1965-1969	63.33	69.60	L&T
	1970-1974	64.92	71.37	L&T
	1975-1979	66.31	73.45	WHO
	1981	68.15	75.19	WHO
Romania	1960-1964	64.83	68.68	WHO
	1965-1969	65.80	70.07	WHO
	1970-1974	66.59	71.05	L&T
	1975-1979	67.25	72.03	WHO
	1980-1983	66.83	72.37	WHO
Scotland	1910-1912	50.11	53.13	C&D
	1920-1922	53.12	56.39	C&D
	1930-1932	55.99	59.51	C&D
	1948	63.92	67.76	C&D
	1952	65.24	69.81	C&D
	1959	66.10	71.72	C&D
	1960-1964	66.27	72.16	L&T
	1965-1969	66.99	73.18	L&T
	1970-1974	67.34	73.74	L&T
United Kingdom:				
Scotland	1975-1979	68.07	74.41	WHO
	1980-1983	69.17	75.40	WHO
Spain	1900	33.87	35.71	C&D
	1910	40.94	42.57	C&D
	1920	40.26	42.07	C&D
	1940	47.14	53.32	C&D
	1965-1969	68.64	73.92	L&T
	1970-1974	69.72	75.28	L&T
	1975-1979	70.93	76.88	WHO
Sweden	1851-1860	40.52	44.42	C&D
	1861-1870	42.83	46.44	C&D
	1871-1880	45.31	48.66	C&D
	1881-1890	48.61	51.54	C&D
	1931-1940	63.86	66.20	C&D
	1941-1945	67.14	69.81	C&D
	1946-1950	69.18	71.70	C&D
	1951-1955	70.65	73.55	C&D
	1959	71.87	75.38	C&D
	1960-1964	71.54	75.49	L&T
	1965-1969	71.82	76.46	L&T
	1970-1974	72.17	77.63	L&T
	1975-1979	72.38	78.42	WHO
	1980-1983	73.26	79.32	WHO
Switzerland	1960-1964	68.77	74.49	L&T
	1965-1969	69.69	75.48	L&T
	1970-1974	70.65	76.83	L&T
	1975-1979	71.93	78.56	WHO
	1980-1982	72.59	79.22	WHO
United States	1900-1902	48.29	51.14	C&D
	1909-1911	50.29	53.69	C&D
	1919-1921	56.41	58.59	C&D
	1929-1931	59.19	62.75	C&D
	1947-1950	66.03	71.70	C&D[g]
	1955-1958	67.48	74.03	C&D[h]
	1960-1964	66.87	73.53	L&T
	1965-1969	66.86	74.18	L&T
	1970-1974	67.57	75.24	L&T
	1975-1979	69.48	77.17	WHO
	1980-1982	70.51	77.85	WHO
Yugoslavia	1960-1964	62.83	66.07	L&T
	1965-1969	64.97	68.91	L&T
	1970-1974	65.94	70.75	L&T
	1975-1979	67.43	72.52	WHO
	1980-1981	67.69	73.23	WHO

[a] *Sources*:

C&D. Input life tables for the Coale and Demeny (1966) model life table system.

L&T. United Nations (1982). *Levels and Trends of Mortality Since 1950* (Sales No. E.81.XIII.3), table IIA.2, pp. 69-82.

WHO. Based on registered deaths and estimated populations by age and sex provided by the World Health Organization on magnetic tape.

[b] Average of life tables for 1956 and 1958.

[c] Average of life tables for 1948 and 1951.

[d] Average of life tables for 1958 and 1959.

[e] Average of life tables for 1955 and 1956-1957.

[f] Average of life tables for 1955-1956 and 1957-1958.

[g] Average of life tables for 1947 and 1950.

[h] Average of life tables for 1955 and 1958.

TABLE 3. RESIDUAL OF REGRESSION OF THE FEMALE-MALE DIFFERENCE ON BOTH SEXES LIFE EXPECTANCY AT BIRTH: LESS DEVELOPED COUNTRIES

Country and region	Reference period	Regression residual[a]
Africa		
Northern Africa		−0.71
Algeria	1969-1971	−1.73
Egypt	1965-1967	−0.36
Egypt	1975-1977	0.43
Morocco	1972	−0.71
Tunisia	1968-1969	−2.17
Other Africa		3.00
Burundi	1970-1971	0.80
Cameroon	1958-1965	4.72
Liberia	1970-1971	2.30
Mauritius	1961-1963	−0.10
Mauritius	1971-1973	0.84
Réunion	1961-1962	4.25
Réunion	1967-1968	3.70
Réunion	1970	4.75
Americas		
Temperate South America		1.78
Argentina	1959-1961	1.72
Argentina	1969-1970	2.95
Argentina	1979	1.78
Chile	1959-1961	2.49
Chile	1969-1971	2.25
Uruguay	1962-1964	0.87
Uruguay	1974-1976	1.66
Other America		0.02
Colombia	1963-1965	−0.38
Costa Rica	1962-1964	−0.93
Costa Rica	1972-1974	−1.73
Cuba	1969-1971	−2.45
El Salvador	1970-1972	2.19
Guadeloupe	1966-1968	0.90
Guadeloupe	1981-1983	1.19
Guatemala	1963-1965	0.28
Guatemala	1972-1973	−0.56
Guyana	1959-1961	0.11
Guyana	1969-1971	0.37
Honduras	1960-1962	3.66
Honduras	1973-1975	2.30
Jamaica	1959-1961	0.01
Jamaica	1969-1971	−0.81
Martinique	1960-1962	−0.37
Martinique	1966-1968	0.43
Martinique	1981-1983	−1.72
Mexico	1969-1971	0.56
Panama	1960-1970	−1.43
Panama	1970-1980	−2.94
Puerto Rico	1949-1951	−0.80
Puerto Rico	1959-1961	−0.62
Puerto Rico	1969-1971	0.42
Puerto Rico	1979-1981	1.09
Suriname	1963-1964	0.02
Trinidad and Tobago	1945-1947	0.52
Trinidad and Tobago	1959-1961	−0.11
Trinidad and Tobago	1969-1971	−0.34
Venezuela	1970-1972	0.87
Asia		
Southern Asia		−2.86
Bangladesh	1964-1965	−3.50
Bangladesh	1981	−2.81
India	1971-1976	−2.42
Iran	1973-1976	−3.42
Nepal	1974-1976	−2.86
Pakistan	1962-1965	−2.43
Pakistan	1968-1971	−4.37
Sri Lanka	1945-1947	−1.92
Sri Lanka	1952-1954	−4.09
Sri Lanka	1962-1964	−3.43
Sri Lanka	1970-1972	−1.60
East Asia and Southeast Asia		0.99
Hong Kong	1960-1962	2.42
Hong Kong	1970-1972	2.65
Hong Kong	1976	0.83
Hong Kong	1981	−0.57
Malaysia (Peninsular)	1969-1971	−0.01
Philippines	1969-1971	1.62
Philippines	1974-1976	0.84
Republic of Korea	1971-1975	2.86
Singapore	1956-1958	2.01
Singapore	1969-1971	0.86
Singapore	1979-1981	−0.35
Thailand	1969-1971	1.12
Western South Asia		−2.64
Israel	1960-1962	−3.65
Israel	1971-1973	−2.64
Kuwait	1974-1976	−0.67
Kuwait	1979-1981	−1.65
Syrian Arab Republic	1976-1978	−3.00

[a] Negative (positive) residuals (E_i) indicate relatively high female (male) mortality. The underlined figure following the region heading is the median residual for that region.

SEX DIFFERENTIALS IN LIFE EXPECTANCY AND MORTALITY IN DEVELOPED COUNTRIES: AN ANALYSIS BY AGE GROUPS AND CAUSES OF DEATH FROM RECENT AND HISTORICAL DATA

United Nations Secretariat *

SUMMARY

In the early 1980s a newborn girl in the developed countries could expect to live many years longer than a boy. The number of additional years of female life expectancy ranged from five to nine years in most of the countries. These large sex differentials in life expectancy reflect the fact that males in developed countries today have higher mortality than females in every age group and for most causes of death. In contrast, early in the twentieth century higher female than male death rates in many age groups were not uncommon, and sex differentials in life expectancy were narrower by several years in most developed countries.

The first half of the paper presents estimates for the early 1980s of the size of sex differentials in life expectancy in developed countries, and the contributions of age groups and causes of death to those differentials. Most of the developed countries are included in the analysis. Diseases of the circulatory system were found to account for nearly 40 per cent of the mean sex differential in life expectancy; neoplasms for 18 per cent; and accidents, suicide and violence for 19 per cent.

The second half of the paper examines trends in sex differentials in life expectancy since the late nineteenth or early twentieth centuries, in the context of the transition from high to low mortality. The contributions of age groups and causes of death to changes in the sex differentials between around 1900 and the 1980s are estimated for selected countries.

INTRODUCTION

In the early 1980s, females in most developed countries outlived males by between five and nine years. Such large sex differences in life expectancy in the absence of war are a relatively recent phenomenon in the demographic history of developed countries. To take two examples, in England and Wales females outlived males by 1.6 years in 1838-1844, but the differential had grown to 6.0 years by 1982-1984 (United Kingdom, 1986, p. 25). In Japan, females outlived males by only 0.6 year in 1900, but by 5.5 years in 1982 (Nanjo and Kobayashi, 1985, tables B2 and B4).

The widening of the sex differential in life expectancy has been one of the features of twentieth century mortality trends in developed countries. It has occurred in the context of other rather remarkable changes in mortality conditions: life expectancy at birth has been greatly extended, the ages at which most people die have shifted from the very young to the very old, and heart disease and cancer have replaced the infectious and parasitic diseases as the major causes of death.

The present paper examines levels and trends in the sex differential in life expectancy and identifies the age groups and the causes of death responsible for the sex differentials. Although the emphasis is on the early 1980s, long-term trends since the nineteenth or early twentieth centuries are also examined for selected countries. Section I of the paper presents some introductory remarks on the data base and on the measures of sex differentials used for the study. Section II presents data for the early 1980s on the size of the sex differentials in life expectancy in most of the developed countries, and on the contribution of 10 age groups and the major causes of death to the differentials. Section III examines long-term trends in sex differentials in life expectancy in several countries, and presents estimates of the contribution of age groups and causes of death to the changing sex differential. Section IV summarizes the main conclusions of the paper.

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I. DATA BASE, MEASURES EMPLOYED, QUALITY AND COMPARABILITY OF THE DATA

A. *Data base*

The data base for the study consists of a set of life tables covering the period from the nineteenth century to the early 1980s. For the period prior to 1950, the life tables are mainly from two sources. The first is *Causes*

of Death: Life Tables for National Populations (Preston, Keyfitz and Schoen, 1972). The life tables from this source are used for the analysis of sex differentials in mortality by age group and by causes of death. The second source for the period prior to 1950 is the life tables collected by Coale and Demeny, from which they constructed their regional model life tables (Coale and Demeny, 1966). These life tables do not provide information on causes of death, but they have been used, together with the life tables from Preston, Keyfitz and Schoen, for determining trends in sex differentials in life expectancy and their decomposition by age group.

For the 1950s to the 1980s, life tables were constructed by the Population Division of the Department of International Economic and Social Affairs, United Nations Secretariat, based mainly on the mortality and population data banks of the World Health Organization (WHO). The WHO mortality file includes data on registered deaths by sex, age and cause, reported annually by countries to WHO.[1] National statistical publications were also consulted, as were various issues of the *Demographic Yearbook,* published by the Statistical Office of the United Nations Secretariat.

B. *Measures employed in the analysis*

Life expectancy at birth is a measure that summarizes mortality at all ages in a population. Because it is not affected by the age distribution of the population, it is suitable for making comparisons among populations with different age structures. The difference between the male and female life expectancy at birth is the measure of the overall sex differential in mortality that has been used most frequently in the analysis.

In sections II and III, sex differentials in life expectancy at birth have been decomposed into age components that indicate the contributions of sex differences in mortality in specified age groups to the overall sex differential in life expectancy. Because of the interrelationships among the functions of the life table, the contribution of a given age group to the sex differential in life expectancy is determined by the difference between the male and female death rates in the age group (the larger the difference, the greater the contribution of the age group); the location of the age group along the age continuum (the younger the age group, the greater the contribution of a given difference in death rates to the difference in life expectancy); and mortality in all the preceding age groups, for such antecedent mortality determines the number of survivors to the age group in question.[2]

Sex ratios of age-specific death rates (computed as the male death rate divided by the female death rate) are useful indicators of the mortality disadvantage of one sex relative to the other by age group. In the developed countries, the highest sex ratios of death rates are generally at ages 15-24: the average sex ratio in this age group in the developed countries in the early 1980s was 2.7 (see table 3), indicating highly adverse mortality conditions for males. However, the sex ratio of death rates in an age group is not in itself a good indicator of the contribution of that age group to the sex differential in life expectancy. This can be seen in the following data for five countries with the same sex ratio of death rates (2.8) at ages 15-24, but with contributions to the sex differential in life expectancy that range from 0.3 year to 0.6 year.

		For ages 15-24		
Country	*Year*	*Sex ratio of death rate*	*Sex difference in death rates (per 1,000)*	*Contribution of age group to the sex differential in e_0 (years)*
Scotland (UK)	1980-1983	2.8	0.6	0.3
Czechoslovakia	1980-1982	2.8	0.7	0.4
Italy	1980	2.8	0.7	0.4
Belgium	1980-1981	2.8	0.9	0.5
United States	1980-1982	2.8	1.0	0.6

Source: Tables 3 and 4 and life tables constructed for the study (see table 2, sources).

The relationships between sex ratios of death rates, sex differences in death rates and the contribution of an age group to the sex differential in life expectancy are illustrated in longitudinal data for England and Wales for infants and for children aged 1-4 years (table 1). Between 1891-1900 and 1980-1983, infant mortality declined by over 90 per cent, but the sex ratio of the infant death rates has remained remarkably constant, increasing slightly from 1.22 to 1.29. Despite the increase in the sex ratio, however, the contribution of this age group to the overall sex difference in life expectancy declined from 1.69 years in 1891-1900 to only 0.21 year in 1980-1983. The large contribution at the earlier period resulted from the large absolute difference between the male and female infant death rates (31.1 per 1,000), which was in turn related to the high infant mortality rates that prevailed (171.7 and 140.6 per 1,000 for males and females, respectively). The data for ages 1-4 show similar relationships, although the magnitudes are different because of the low death rates at ages 1-4 compared with those of infancy.

The data in table 1 illustrate the phenomenon of "level" effects described by Preston, whereby sex differences in death rates tend to contract as mortality levels decline, but sex ratios of death rates tend to increase. "This type of situation is encountered *most of the time* when comparisons are made between a high mortality population and a low mortality population" (Preston, 1976, p. 125).

C. *Data quality and comparability*

The developed countries generally have well-functioning vital registration systems, with timely and virtually complete registration of births and deaths. For countries that report data on medical certification of cause of death, a relatively high proportion of deaths are medically certified in most of these countries (United Nations, 1982a, table 31).

The main problems in cause-of-death analysis in the developed countries concern comparability among countries in classifying deaths by cause, as well as comparability over time within countries, because of periodic revisions of the International Classification of Diseases and changes in diagnostic preferences (Hansluwka, 1986). The causes of death to be entered on the death

TABLE 1. COMPARISON OF TWO MEASURES OF SEX DIFFERENTIALS IN MORTALITY, ENGLAND AND WALES, 1891-1900 TO 1980-1983

Period	Death rates (per 1,000 male (female) live births for age 0; per 1,000 male (female) population for age 1-4)			Sex ratios of death rates (males/females)	Contribution of age group to the sex differential in life expectancy at birth (years)
	Males (1)	Females (2)	Difference (males minus females) (3)	(4)	(5)
			Infants (age 0)		
1891-1900	171.7	140.6	31.1	1.22	1.69
1901-1910	144.2	117.4	26.8	1.23	1.55
1930-1932	71.9	54.5	17.4	1.32	1.12
1950-1954	31.4	24.3	7.0	1.29	0.51
1970-1974	19.5	15.0	4.5	1.30	0.33
1980-1983	12.4	9.6	2.8	1.29	0.21
			1-4 years		
1891-1900	25.0	23.8	1.2	1.05	0.20
1901-1910	18.9	18.0	0.9	1.05	0.17
1930-1932	7.5	6.8	0.7	1.11	0.17
1950-1954	1.3	1.1	0.2	1.19	0.06
1970-1974	0.8	0.6	0.1	1.20	0.04
1980-1983	0.5	0.4	0.1	1.21	0.03

Source: Based on country life tables for dates before 1950; for 1950-1954, 1970-1974 and 1980-1983, based on life tables calculated by the United Nations from the World Health Organization data bank.

Note: Because of rounding, the difference between the male and female rates in columns (1) and (2) may differ from the values shown in column (3).

certificate are "all those diseases, morbid conditions or injuries which either resulted in or contributed to death and the circumstances of the accident or violence which produced such injuries" (World Health Organization, 1977, p. 699). In cases where two or more conditions are recorded on the death certificate, the underlying cause is to be selected for single-cause tabulation, this being defined as "(*a*) the disease or injury which initiated the train of morbid events leading directly to death, or (*b*) the circumstances of the accident or violence which produced the fatal injury" (World Health Organization, 1977, pp. 699-703 and 763-765). National variations in medical concepts, diagnostic practices and the interpretation of rules for determining the underlying cause of death by coders when the information entered on death certificates is ambiguous or incomplete can be a source of non-comparability among countries.

The farther back in time one goes, the less reliable the mortality data tend to be. Among the data problems encountered are incomplete death registration (particularly of infants); errors in age reporting; inaccurate diagnoses and classification of causes of death; and large percentages of deaths not classified by cause. In section III of this paper, data for the nineteenth and early twentieth centuries are presented for selected countries. These data are based on national life tables by causes of death prepared by Preston, Keyfitz and Schoen (1972), who subjected the data to careful scrutiny for errors and inconsistencies. An important source of error, the inaccurate or inconsistent diagnosis and coding of cause of death, was to some degree overcome by defining broad categories of causes of death that included causes that were likely to be mistaken for one another. The neoplasms category, for example, includes malignant and benign neoplasms and those of an unspecified nature. Despite the inevitable data problems, the authors concluded that the fundamental changes in the structure of mortality in the past century had not been obscured. A second serious problem is less easily overcome. This is the large percentage of deaths assigned to categories of senility, symptoms and ill-defined conditions in the early data, with the result that the deaths in the remaining cause categories are correspondingly understated. As the proportion of deaths not classified by cause has decreased over time because of improvements in death certification, the comparability of death trend data for specific diseases within countries has been affected.

II. THE EARLY 1980s

The developed countries, according to the classification employed by the Population Division of the United Nations Secretariat, include Canada and the United States of America, Japan, all the countries of Europe, Australia and New Zealand, and the Union of Soviet Socialist Republics. Some data for most of these countries have been included in the present section. A few countries have been excluded, either because of lack of recent information (Albania) or small population size (Iceland, Luxembourg and Malta).

A. *Sex differentials in life expectancy*

In the developed countries, male age-specific death rates for all causes combined are almost invariably higher than those of females; consequently, life expectancy at birth is substantially lower for males. The sex differential in life expectancy in developed countries is often wider than differentials between subgroups of the

population classified by some other characteristic. In the United States, for example, life expectancy at birth in 1984 was 7.0 years longer for females than for males, compared with a differential of 5.6 years between the white and black populations (United Nations, 1986, p. 12).

Sex differentials in life expectancy at birth in the early 1980s are shown in table 2 for 31 countries ranked according to size of the differential. The average difference was 6.7 years, with a range from 4.6 years in Greece to 9 years in the USSR.

A given sex differential in life expectancy can result from various combinations of male and female life expectancies. This can be seen from the country data in table 2, columns (2), (3) and (4). In Yugoslavia and Japan, for example, the size of the sex differential in life expectancy is about 5.6 years. In Yugoslavia, this difference arises from male life expectancy of 67.7 years and female life expectancy of 73.3 years, while in Japan the corresponding values are 74.1 years for males and 79.8 years for females.

Table 2 also shows, in column (5), each country's deviation from the mean sex differential in life expectancy of 6.68 years. A negative sign signifies that the sex differential in life expectancy is below the mean, while a positive sign indicates the opposite. The amounts in column (5) of table 2 have been decomposed into four components (two for each sex), which indicate how much

TABLE 2. LIFE EXPECTANCY AT BIRTH, THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AND COUNTRY DEVIATIONS FROM THE MEAN SEX DIFFERNTIAL, DEVELOPED COUNTRIES RANKED BY SIZE OF THE SEX DIFFERENTIAL, EARLY 1980S OR LATEST AVAILABLE PERIOD

(Years)

Country	Period	Rank[a]	Life expectancy at birth				Country deviations from mean differential[c] ((4)-6.68) (5)	Components of the deviation from the mean sex differential in life expectancy			
			Both sexes[b] (1)	Males (2)	Females (3)	Difference ((3)-(2)) (4)		Male e_0 above mean (69.99-(2)) (6)	Male e_0 below mean (69.99-(2)) (7)	Female e_0 above mean ((3)-76.67) (8)	Female e_0 below mean ((3)-76.67) (9)
Greece	1980-1982	1	75.50	73.21	77.80	4.59	−2.09	−3.22	—	1.13	—
Romania	1980-1983	2	69.55	66.81	72.29	5.49	−1.19	—	3.19	—	−4.38
Bulgaria	1980-1982	3	71.36	68.61	74.11	5.51	−1.17	—	1.38	—	−2.56
Ireland	1980-1981	4	72.74	69.98	75.49	5.51	−1.17	—	0.01	—	−1.18
Yugoslavia	1980-1981	5	70.54	67.75	73.32	5.57	−1.11	—	2.24	—	−3.35
Japan	1980-1983	6	76.94	74.13	79.76	5.63	−1.05	−4.13	—	3.08	—
German Democratic Republic	1980-1983	7	72.02	69.04	74.99	5.95	−0.73	—	0.95	—	−1.68
Spain	1975-1979	8	74.05	71.08	77.03	5.95	−0.73	−1.08	—	0.35	—
England and Wales (UK)	1980-1983	9	74.17	71.18	77.16	5.99	−0.69	−1.18	—	0.49	—
Denmark	1980-1983	10	74.52	71.44	77.59	6.15	−0.53	−1.45	—	0.92	—
New Zealand	1980-1983	11	73.55	70.48	76.63	6.15	−0.53	−0.48	—	—	−0.05
Sweden	1980-1983	12	76.34	73.26	79.41	6.16	−0.52	−3.26	—	2.74	—
Scotland (UK)	1980-1983	13	72.27	69.17	75.37	6.20	−0.48	—	0.82	—	−1.30
Northern Ireland (UK)	1980-1982	14	72.42	69.28	75.56	6.28	−0.40	—	0.71	—	−1.11
Italy	1980	15	74.32	70.97	77.66	6.68	0.00	−0.98	—	0.98	—
Switzerland	1980-1982	16	75.96	72.59	79.33	6.74	0.06	−2.60	—	2.66	—
Germany, Federal Republic of	1980-1983	17	73.72	70.34	77.09	6.74	0.06	−0.35	—	0.41	—
Netherlands	1980-1983	18	76.18	72.76	79.61	6.85	0.17	−2.77	—	2.94	—
Belgium	1980-1981	19	73.38	69.94	76.82	6.88	0.20	—	0.06	0.14	—
Norway	1980-1983	20	76.11	72.64	79.58	6.94	0.26	−2.65	—	2.91	—
Portugal	1981	21	71.70	68.18	75.23	7.06	0.38	—	1.82	—	−1.44
Australia	1980-1982	22	74.81	71.25	78.37	7.12	0.44	−1.26	—	1.70	—
Austria	1980-1983	23	72.86	69.28	76.45	7.17	0.49	—	0.71	—	−0.22
Canada	1980-1982	24	75.43	71.80	79.06	7.26	0.58	−1.81	—	2.39	—
Czechoslovakia	1980-1982	25	70.69	67.00	74.38	7.38	0.70	—	3.00	—	−2.30
United States	1980-1982	26	74.28	70.52	78.05	7.53	0.85	−0.53	—	1.38	—
Hungary	1980-1983	27	69.22	65.42	73.02	7.60	0.92	—	4.58	—	−3.69
Poland	1980-1983	28	71.03	66.91	75.15	8.24	1.56	—	3.08	—	−1.52
France	1980-1982	29	75.10	70.97	79.22	8.25	1.57	−0.98	—	2.55	—
Finland	1980-1983	30	74.11	69.84	78.39	8.55	1.87	—	0.16	1.71	—
USSR	1985/86	31	69.00	64.00	73.00	9.00	2.32	—	5.99	—	−3.67
Means, 31 countries			73.35	69.99	76.67	6.68					

Sources: Except for the USSR, based on a set of life tables calculated by the Population Division of the United Nations Secretariat, mainly from registered deaths and population estimates in the World Health Organization data bank. Various issues of the *Demographic Yearbook* (United Nations publication) and official national publications were also consulted. For the USSR, values from the official life table for 1985/86 are shown.

NOTE: This table is patterned after table 3 of A. D. Lopez, "The sex mortality differential in developed countries", in *Sex Differentials in Mortality: Trends, Determinants and Consequences*, A. D. Lopez and L. T. Ruzicka, eds. (Canberra, Australian National University, 1983, pp. 66-67.

[a] Ranked according to size of sex differential in life expectancy (column (4)).

[b] Values for both sexes are unweighted averages of male and female values shown in columns (2) and (3).

[c] Sum of columns (6), (7), (8) and (9).

of the deviation from the mean is attributable to higher than average or lower than average male and female life expectancy. (The mean life expectancy values are 70.0 years for males and 76.7 years for females.) The results of the decomposition are shown in columns (6) through (9). Negative values in columns (6) through (9) signify that the deviation from the mean male or female life expectancy has the effect of narrowing the sex differential in relation to the mean, while positive deviations have the effect of widening the differential.

In general, countries in which life expectancy is above average for one sex will also have above average life expectancy for the other sex. Such countries will have values in columns (6) and (8) of table 2. The converse is also true—countries with lower than average life expectancy for one sex will also tend to show below average life expectancy for the other. For those countries, values will appear in columns (7) and (9). There are very few exceptions to this observation.

Taking the examples of Yugoslavia and Japan again, in both countries the sex differential in life expectancy at birth of 5.6 years is 1.1 year below the mean of 6.7 years for all countries (column (5)). In the case of Yugoslavia, this deviation from the mean value results from male life expectancy that is 2.2 years below the mean male life expectancy (column (7)) and female life expectancy that is 3.3 years below the mean female life expectancy (column (9)). In Japan, in contrast, the 1.1 year deviation results from male life expectancy that is 4.1 years above the male average (column (6)) and female life expectancy that is 3.1 years above the female average (column (8)).

The pattern closest to the mean pattern is that of the Federal Republic of Germany, where both male and female life expectancy are 0.4 year above the mean, and the resulting sex differential is the same as the mean (6.7 years). Among the five countries with the smallest sex differentials in life expectancy (Greece, Romania, Bulgaria, Ireland and Yugoslavia), the deviations from the mean are, in all cases but Greece, due to much lower than average female life expectancy. The patterns among the five countries with the largest sex differentials are more varied: in Hungary, Poland and the USSR, male life expectancy is several years below the average; in Finland, male life expectancy is 0.2 year below average, while female life expectancy is 1.7 years above average; a third pattern is seen in France, where male life expectancy is 1.0 year above average and female life expectancy is 2.6 years above average.

The country data on life expectancy at birth and the sex differential in life expectancy do not show a relationship between the two variables (see figure I). The only apparent pattern shown by these data is a geographic one: most of the countries at both extremes of the distribution by size of the sex differential are in Eastern Europe or Southern Europe.

Sex differentials in mortality by age group are summarized in tables 3, 4 and 5, which present data for 30 or 31 populations for the early 1980s. The pattern of higher male mortality is so pervasive in the developed countries that there is only one instance of higher female mortality in the data as grouped in these tables (the exception was in Belgium at ages 1-4).

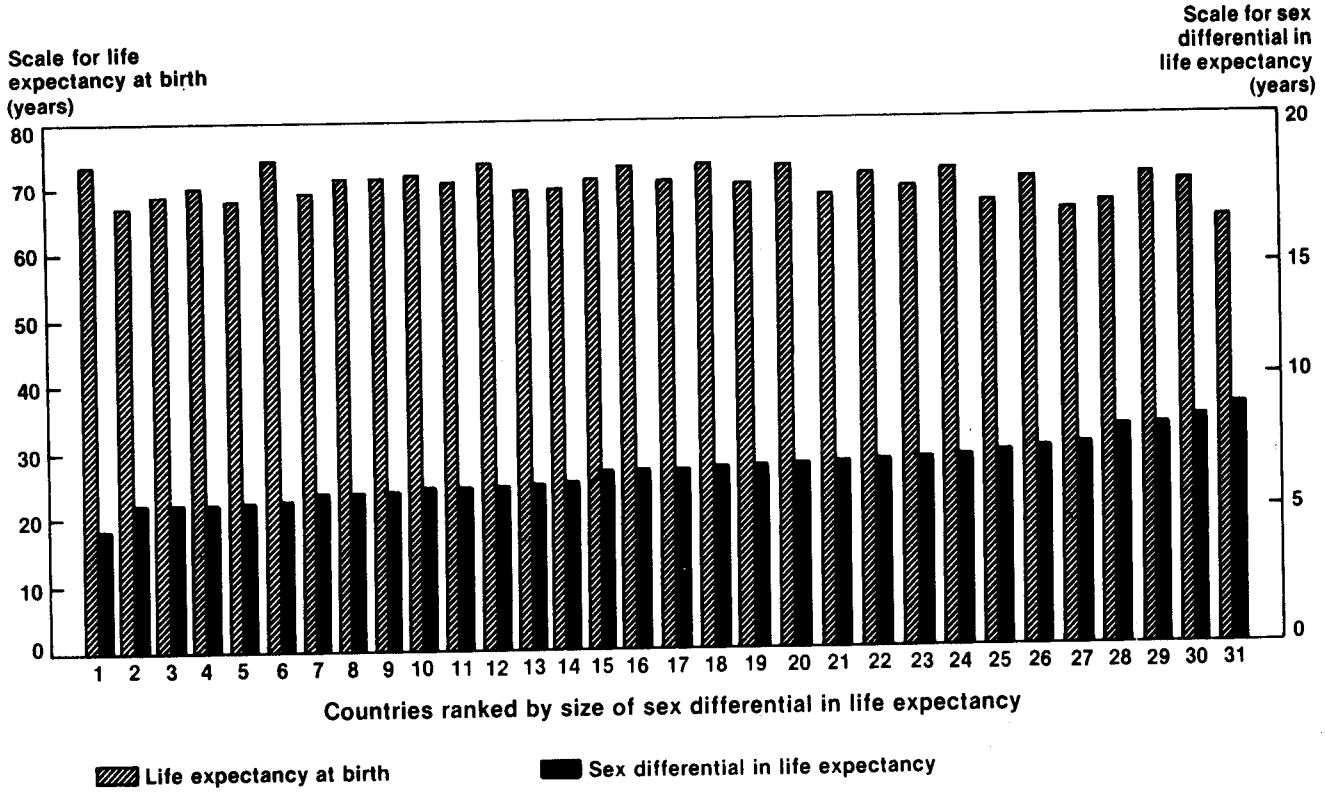
B. *Sex ratios of death rates by age group*

Sex ratios of death rates (table 3) are important as a reflection of disparities, or unusual patterns, in the mortality conditions of males and females. For brevity, table 3 shows ratios for 10-year age groups, but figure II and the discussion that follows considers five-year age groups. Two major patterns of sex ratios of death rates are seen in the developed countries. The most common, referred to here as the "Western", is one of gradually increasing ratios between ages 1-4 and 10-14, followed by sharp increases at ages 15-19 and again at ages 20-24, when sex ratios are at their peak in most countries. The ratios then decline sharply, and reach a low point sometime between ages 35 and 50. The ratios then increase again, but to a level that is usually substantially below that of ages 20-24. The elevated ratios of late middle age are, in many countries, sustained across several age groups, and form a plateau or a hump rather than a second peak. The ratios then drop off steeply after age 60 or 65. Most countries exhibit some variation of this pattern, although differing as to height of peaks, depth of troughs and the ages at which the peaks and troughs occur. The only substantial deviations from the pattern described are in several countries of Eastern and Southern Europe, and the USSR, in which there is a peak ratio at ages 20-24 or 25-29, but no secondary increase in the ratios in middle age. This is the "Eastern" pattern.

The Western and Eastern patterns are depicted in parts *a* and *b*, respectively, of figure II. Parts *c* through *l* show curves of sex ratios of death rates for individual countries grouped by similarities in the shapes of their curves. In the Western pattern, which is based on averages for 22 countries, the sex ratio of death rates increases from 1.3 at age 0 and age 1-4, to a high of 3.0 at age 20-24, then declines sharply to 1.8 at ages 35-39 and 40-44. The ratio then increases gradually to 2.1 at ages 55-59 and 60-64, after which it declines, and reaches 1.2 at ages 85 and over. The countries included in these averages were all the countries of Northern and Western Europe, the countries of Southern Europe, except for Portugal and Yugoslavia, and the developed countries outside Europe (Canada, United States, Japan, Australia and New Zealand).

The Eastern pattern (figure II, part *b*) is based on the average ratios for Bulgaria, Czechoslovakia, Hungary, Poland and Yugoslavia. The Soviet Union has a similar pattern (see part *k* of figure II), but was not included in the averages because the available age distribution did not exactly match that of the other countries. The average ratios in part *b* are similar to those of part *a* through ages 20-24. The decline in the sex ratio after ages 20-24, however, is much more gradual in the Eastern countries than in the Western countries, and there is no trough and subsequent rise in the ratio in middle age. The sex ratio declines gradually from 3.0 at ages 20-24 to 2.3 at ages 50-54, then more steeply to reach 1.1 at ages 85 and over. The Eastern ratios are higher than the Western ratios from ages 25-29 through 55-59, after

Figure I. Life expectancy at birth (both sexes), and the sex differential in life expectancy at birth, developed countries ranked according to size of sex differential, early 1980s or latest available period



Source: Table 2. The names of the countries corresponding to the country codes along the X-axis, and the dates, are given in table 2.

which there is a cross-over, and the Eastern ratios are lower for all subsequent age groups.

The German Democratic Republic and Portugal were not included in either the Western or Eastern averages because their sex ratio patterns were intermediate between the two patterns. Romania was also excluded because of its unique pattern (see part *l*), in which an early peak is absent.

The causes of death that account for the different patterns of sex ratios of death rates are suggested by figure III, which shows sex ratios of death rates by cause for two countries with a "Western" pattern (England and Wales and the United States) and two with an "Eastern" pattern (Hungary and Poland). One of the major differences seen in the shapes of the Western and Eastern curves for all causes (figure II, parts *a* and *b*, respectively) was the much more gradual descent of the sex ratios in the Eastern curve after the peak ratio of ages 20-24. This is partly accounted for by the ratios for accidents and violence (figure III, part *b*), which are much higher in the Eastern than in the Western countries, and exert a strong influence on the shape of the curve for all causes combined in the young adult ages, where they make up a large share of all deaths. The high sex ratios from accidents and violence in the Eastern pattern are mainly a result of higher than average male mortality rather than lower than average female mortality.

There are also differences between the Western and Eastern countries in the shapes of the curves for other causes. The sex ratios of death rates for neoplasms (figure III, part *c*) are higher in Hungary and Poland than in England and Wales and the United States between ages 35-39 and 65-69. There is a cross-over at ages 70-74, after which England and Wales and the United States have higher ratios for neoplasms. This difference in the pattern for neoplasms explains the cross-over observed in the Western and Eastern patterns for all causes (figure II). For the respiratory diseases (figure III, part *d*), sex ratios of death rates are higher in Hungary and Poland than in the two Western countries in all age groups but the oldest. The curves for diseases of the circulatory system (part *e*) do not differ consistently between the Western and Eastern countries before ages 60-64. After this age group, however, the ratios are higher in the two Western countries.

TABLE 3. SEX RATIOS OF DEATH RATES IN 10 AGE GROUPS, DEVELOPED COUNTRIES RANKED BY SIZE OF THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, EARLY 1980s OR LATEST AVAILABLE PERIOD

Country	Period	Rank	Sex ratios of death rates (males/females)									
			0	1-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75 and over
Greece	1980-1982	1	1.2	1.3	1.4	2.5	2.1	1.8	1.8	2.0	1.6	1.2
Romania	1980-1983	2	1.2	1.1	1.7	2.0	1.9	2.1	2.0	1.8	1.5	1.2
Bulgaria	1980-1982	3	1.3	1.2	1.5	2.2	2.2	2.2	2.1	1.9	1.5	1.2
Ireland	1980-1981	4	1.2	1.4	1.9	2.7	2.1	1.7	1.7	1.8	1.7	1.5
Yugoslavia	1980-1981	5	1.1	1.1	1.4	2.2	2.4	2.1	2.1	1.9	1.5	1.3
Japan	1980-1983	6	1.2	1.3	1.6	2.6	1.8	1.8	2.1	2.0	1.8	1.5
German Democratic Republic	1980-1983	7	1.4	1.3	1.5	2.6	2.3	1.9	2.0	1.9	1.7	1.4
Spain	1975-1979	8	1.3	1.2	1.5	2.6	2.1	1.9	2.0	2.1	1.9	1.4
England and Wales (UK)	1980-1983	9	1.3	1.2	1.4	2.5	1.8	1.5	1.6	1.9	1.9	1.7
Denmark	1980-1983	10	1.3	1.3	1.3	2.6	2.1	1.5	1.5	1.8	1.9	1.6
New Zealand	1980-1983	11	1.2	1.3	1.4	2.4	2.1	1.5	1.5	1.8	1.8	1.6
Sweden	1980-1983	12	1.1	1.3	1.5	2.4	2.2	1.8	1.9	2.0	2.0	1.6
Scotland (UK)	1980-1983	13	1.3	1.5	1.5	2.8	2.0	1.6	1.7	1.8	1.8	1.6
Northern Ireland (UK)	1980-1982	14	1.3	1.4	1.3	3.0	2.1	1.6	1.8	1.8	1.9	1.6
Italy	1980	15	1.3	1.1	1.4	2.8	2.1	1.9	2.2	2.3	2.0	1.4
Switzerland	1980-1982	16	1.3	1.5	1.6	2.8	2.2	1.8	1.9	2.2	2.1	1.6
Germany, Federal Republic of	1980-1983	17	1.3	1.2	1.5	2.7	2.1	1.9	2.0	2.1	2.0	1.5
Netherlands	1980-1983	18	1.3	1.2	1.5	2.3	1.8	1.5	1.8	2.2	2.3	1.7
Belgium	1980-1981	19	1.4	1.0	1.3	2.8	2.1	1.7	1.9	2.2	2.1	1.6
Norway	1980-1983	20	1.3	1.4	1.8	3.3	2.6	1.8	2.1	2.2	2.1	1.6
Portugal	1981	21	1.2	1.4	1.4	3.7	2.5	2.2	2.2	2.1	1.8	1.4
Australia	1980-1982	22	1.3	1.3	1.7	3.0	2.4	1.8	1.9	2.0	2.0	1.7
Austria	1980-1983	23	1.4	1.3	1.4	3.3	2.5	2.1	2.3	2.2	1.9	1.5
Canada	1980-1982	24	1.3	1.3	1.6	3.1	2.3	1.7	1.9	2.0	2.0	1.7
Czechoslovakia	1980-1982	25	1.4	1.3	1.6	2.8	2.7	2.5	2.4	2.2	1.8	1.4
United States	1980-1982	26	1.2	1.3	1.5	2.8	2.5	1.9	1.8	1.9	1.9	1.7
Hungary	1980-1983	27	1.3	1.4	1.5	2.6	2.4	2.3	2.3	2.1	1.8	1.4
Poland	1980-1983	28	1.3	1.3	1.6	3.2	3.1	2.7	2.5	2.2	1.9	1.5
France	1980-1982	29	1.4	1.3	1.5	2.7	2.4	2.1	2.5	2.6	2.3	1.7
Finland	1980-1983	30	1.2	1.7	1.8	3.2	3.0	2.7	2.9	2.8	2.2	1.6
USSR	1973-1974	31	—1.3—		1.5	2.7	3.3	2.9	2.5	2.3	2.0[a]	1.4[b]
Means, 30 countries[c]			1.3	1.3	1.5	2.7	2.3	1.9	2.0	2.1	1.9	1.5

Sources: Except for the USSR, based on a set of life tables calculated by the Population Division of the United Nations Secretariat for the present study (see table 2 sources). For the USSR, sex ratios have been calculated from published official death rates.

[a] 65-69 years.

[b] 70 years and over.

[c] Excluding USSR.

C. *Contribution of age groups to the sex differential in life expectancy*

The contributions of age groups to the sex differential in life expectancy are shown in tables 4 and 5, and in figure IV, for developed countries in the early 1980s. Table 4 presents contributions of 10 age groups in years, and table 5 in percentages. In figure IV, absolute contributions to the sex differential in life expectancy are presented for broader age groups. Ages below 15 years contributed very little to the sex differential in life expectancy. For infants, contributions ranged from 0.1 year to 0.4 year, with an average of 0.2 year. The countries with the largest contributions for infants (0.3 or 0.4 year) were all in Eastern Europe (Bulgaria, Czechoslovakia, Hungary, Poland and Romania). These countries have higher than average infant mortality rates relative to other developed countries, and larger than average differences between the male and female rates. Most of the countries with the smallest contributions (0.1 year) have very low infant mortality levels (Finland, Japan, Norway and Sweden).

Of the 10 age groups shown, the contribution to the sex differential in life expectancy was smallest at ages 1-4 (0.1 year or less) in all countries. The next age group, 5-14 years, has the lowest mortality levels in all the developed countries. The average contribution from this age group was slightly higher than at ages 1-4, but still below 0.1 year.

Although sex ratios of death rates are highest at ages 15-24 in nearly all of the countries (the average ratio, shown in table 3, is 2.7), this age group generally made only a modest contribution to the sex differential in life expectancy. This is because differences between male and female death rates are still relatively low at ages 15-24. The average contribution was 0.4 year, and ranged from 0.2 year in the Netherlands to 0.8 year in Portugal.

The countries towards the bottom of table 4 are those with the largest sex differentials in life expectancy at birth. Yet, for those countries, the size of the contributions from age groups 0, 1-4 and 5-14 are not significantly higher than for the remaining countries. These young age groups, therefore, are not responsible

Figure II. Sex ratios of death rates by age group, developed countries, early 1980s or latest available period

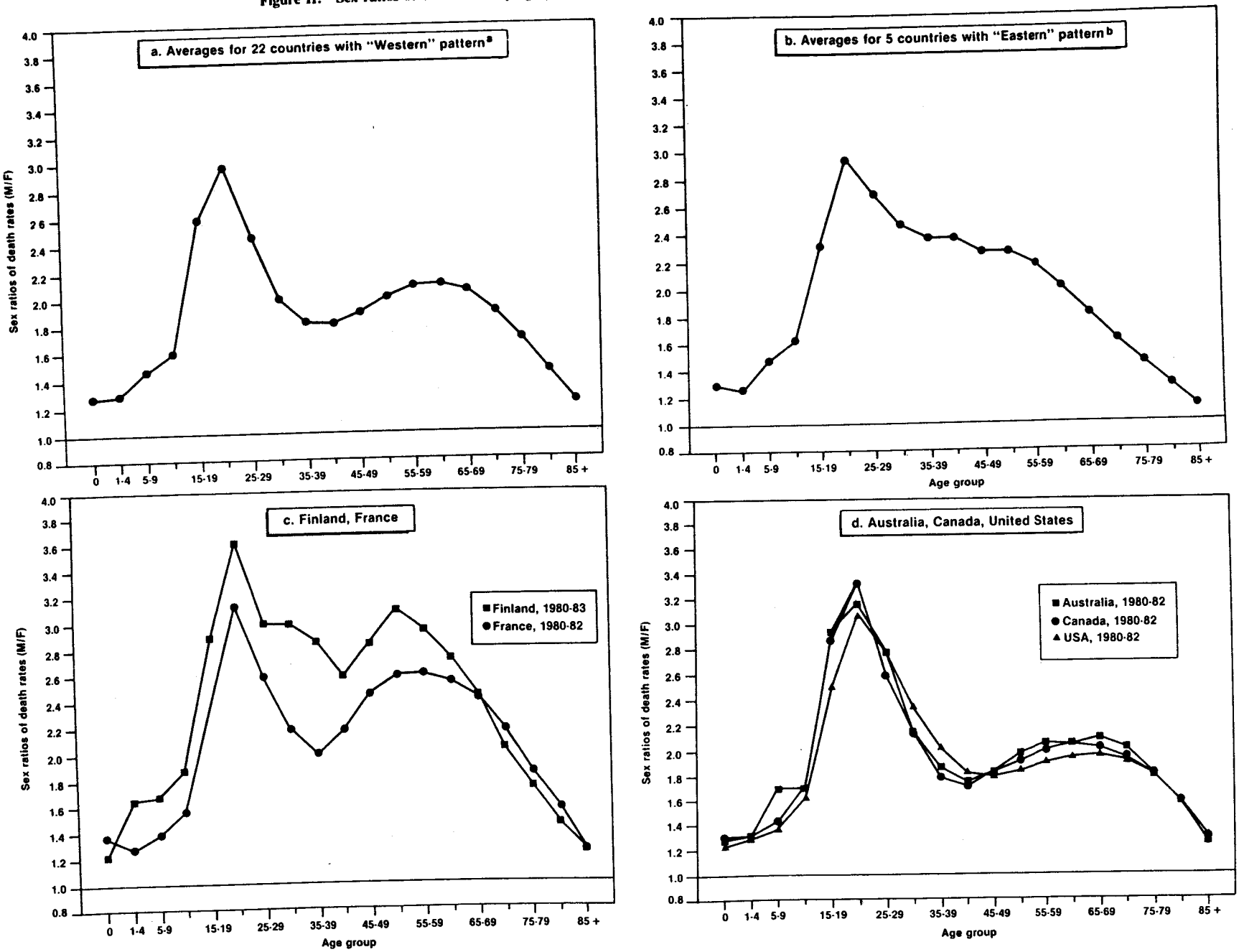
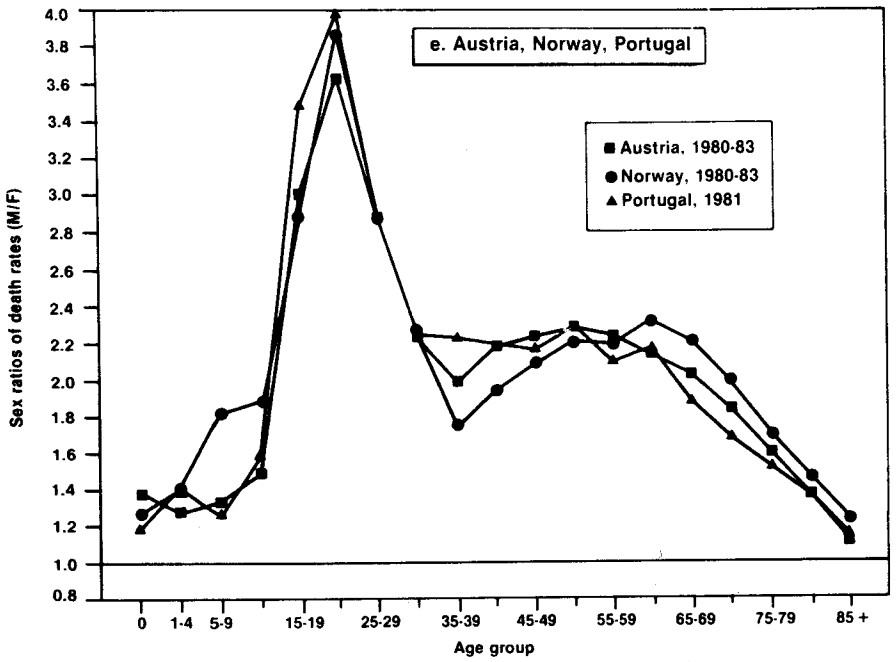
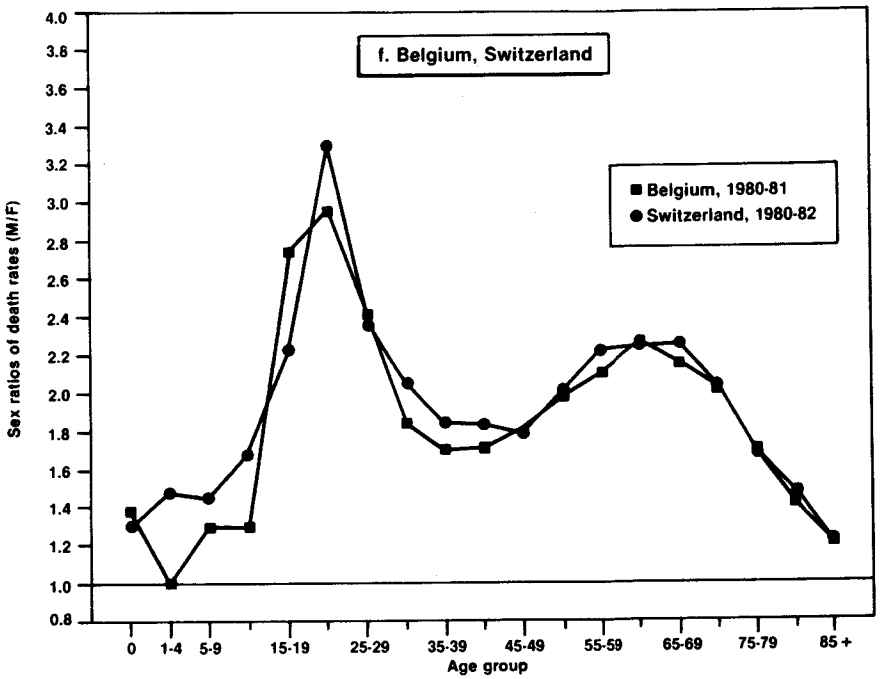
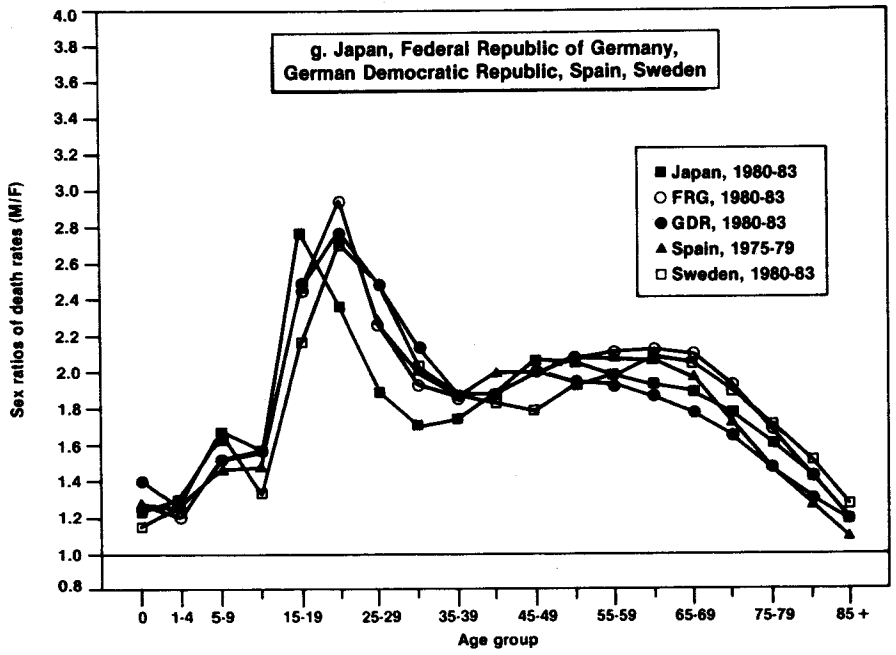


Figure II (*cont.*)







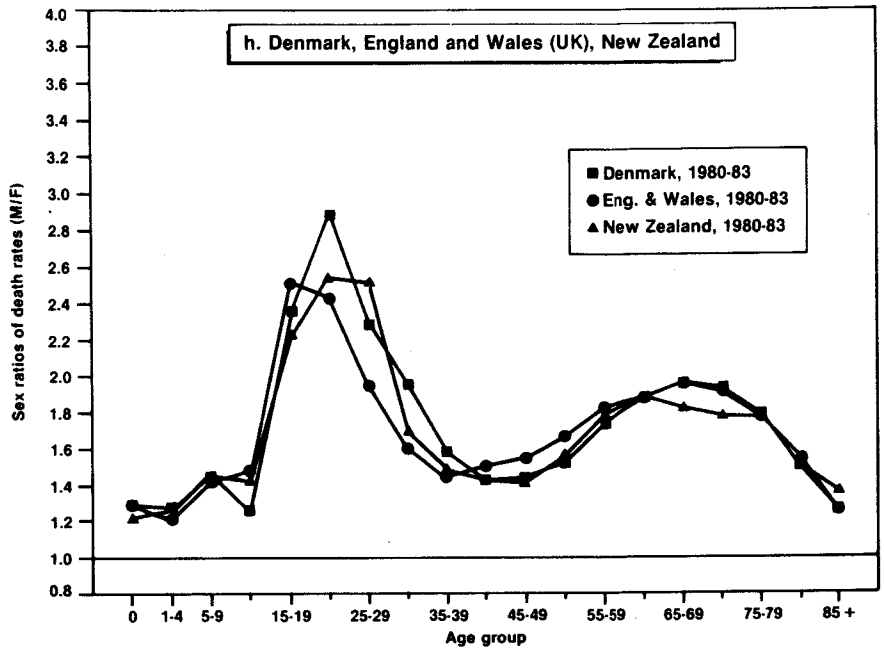
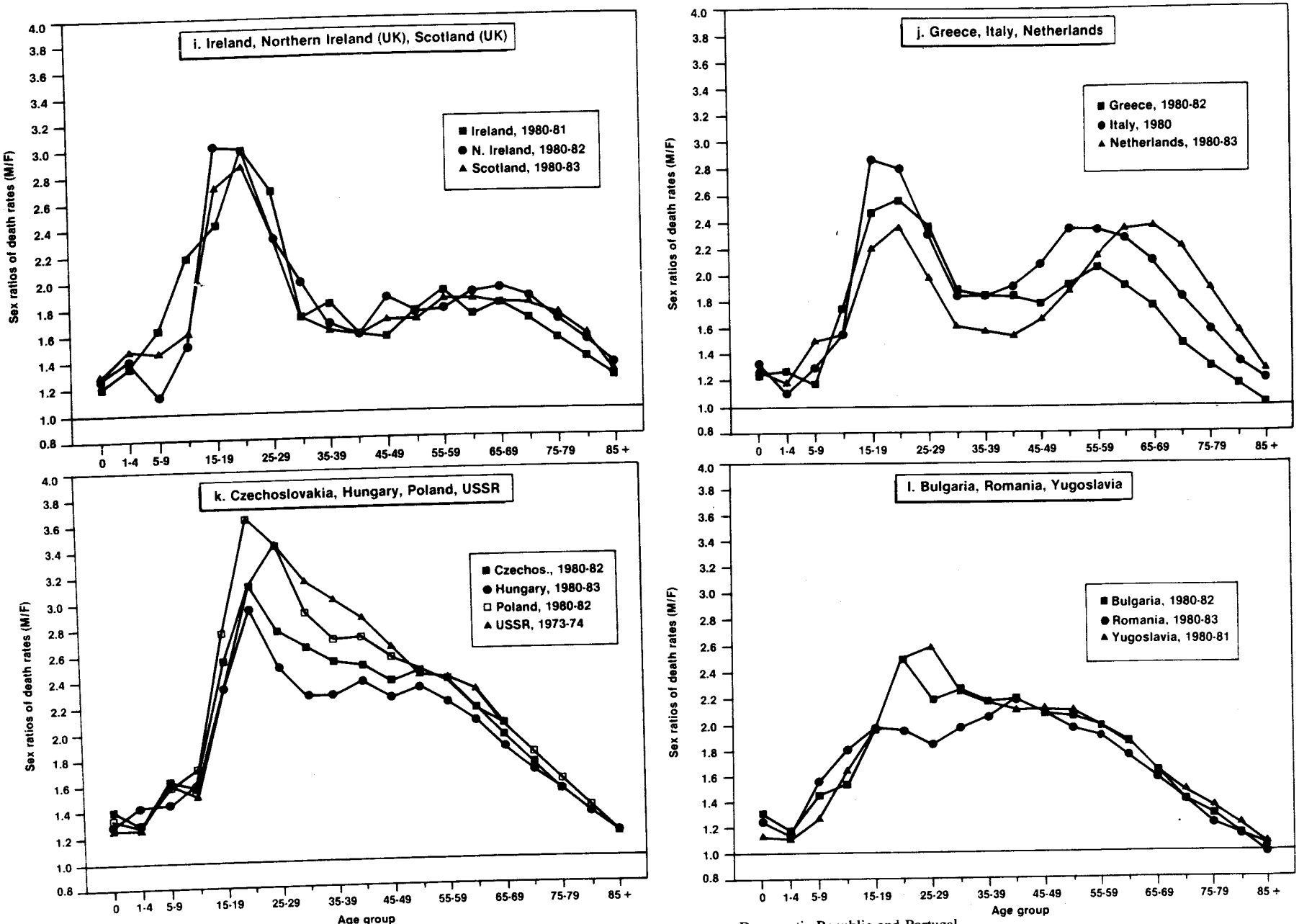


Figure II (*cont.*)



Source: See table 2 sources. For the USSR, official death rates for 1973-1974 were used.

[a] The 22 countries include all the countries shown in parts (c) through (j), except the German Democratic Republic and Portugal.

[b] Bulgaria, Czechoslovakia, Hungary, Poland, Yugoslavia.

TABLE 4. CONTRIBUTION OF 10 AGE GROUPS TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, DEVELOPED COUNTRIES RANKED BY SIZE OF THE SEX DIFFERENTIAL, EARLY 1980S OR LATEST AVAILABLE PERIOD

(Years)

Country	Period	Rank	Sex differential in life expectancy (females minus males) (1)	Contribution of age group to the sex differential in life expectancy: 0 (2)	1-4[a] (3)	5-14[a] (4)	15-24 (5)	25-34 (6)	35-44 (7)	45-54 (8)	55-64 (9)	65-74 (10)	75 and over (11)
Greece	1980-1982	1	4.59	0.3	0.04	0.06	0.3	0.3	0.3	0.5	1.1	1.2	0.6
Romania	1980-1983	2	5.49	0.4	0.06	0.17	0.3	0.4	0.7	1.0	1.2	1.0	0.3
Bulgaria	1980-1982	3	5.51	0.4	0.05	0.10	0.3	0.4	0.6	0.9	1.3	1.1	0.5
Ireland	1980-1981	4	5.51	0.1	0.05	0.11	0.3	0.2	0.3	0.6	1.3	1.5	1.0
Yugoslavia	1980-1981	5	5.57	0.3	0.03	0.08	0.3	0.4	0.6	1.0	1.3	1.1	0.6
Japan	1980-1983	6	5.63	0.1	0.04	0.07	0.3	0.2	0.3	0.7	1.0	1.4	1.4
German Democratic Republic	1980-1983	7	5.95	0.3	0.04	0.08	0.4	0.4	0.4	0.9	1.2	1.4	0.8
Spain	1975-1979	8	5.95	0.3	0.05	0.09	0.3	0.3	0.4	0.8	1.3	1.5	0.9
England and Wales (UK)	1980-1983	9	5.99	0.2	0.03	0.06	0.3	0.2	0.2	0.6	1.3	1.8	1.4
Denmark	1980-1983	10	6.15	0.2	0.03	0.05	0.3	0.3	0.3	0.5	1.2	1.7	1.5
New Zealand	1980-1983	11	6.15	0.2	0.04	0.07	0.5	0.3	0.2	0.5	1.3	1.6	1.5
Sweden	1980-1983	12	6.16	0.1	0.02	0.05	0.3	0.3	0.3	0.7	1.2	1.7	1.6
Scotland (UK)	1980-1983	13	6.20	0.2	0.05	0.07	0.3	0.2	0.3	0.7	1.4	1.6	1.3
Northern Ireland (UK)	1980-1982	14	6.28	0.2	0.06	0.05	0.4	0.3	0.3	0.7	1.3	1.7	1.2
Italy	1980	15	6.68	0.3	0.01	0.07	0.4	0.2	0.4	0.9	1.6	1.7	1.1
Switzerland	1980-1982	16	6.74	0.2	0.06	0.08	0.5	0.3	0.3	0.7	1.4	1.8	1.5
Germany, Federal Republic of	1980-1983	17	6.74	0.2	0.03	0.07	0.4	0.3	0.4	0.9	1.4	1.8	1.2
Netherlands	1980-1983	18	6.85	0.2	0.02	0.07	0.2	0.2	0.2	0.6	1.5	2.1	1.8
Belgium	1980-1981	19	6.88	0.3	0.00	0.05	0.5	0.3	0.4	0.8	1.5	1.9	1.2
Norway	1980-1983	20	6.94	0.1	0.05	0.09	0.4	0.3	0.3	0.8	1.5	1.8	1.5
Portugal	1981	21	7.06	0.3	0.10	0.10	0.8	0.5	0.6	1.0	1.4	1.4	0.9
Australia	1980-1982	22	7.12	0.2	0.04	0.09	0.5	0.4	0.3	0.8	1.4	1.9	1.6
Austria	1980-1983	23	7.17	0.3	0.04	0.06	0.6	0.4	0.5	1.1	1.5	1.6	1.0
Canada	1980-1982	24	7.26	0.2	0.04	0.08	0.5	0.4	0.3	0.8	1.4	1.8	1.8
Czechoslovakia	1980-1982	25	7.38	0.4	0.04	0.09	0.4	0.4	0.7	1.2	1.8	1.6	0.8
United States	1980-1982	26	7.53	0.2	0.04	0.07	0.6	0.5	0.5	0.8	1.4	1.7	1.7
Hungary	1980-1983	27	7.60	0.4	0.05	0.08	0.4	0.5	0.9	1.4	1.7	1.4	0.8
Poland	1980-1983	28	8.24	0.4	0.05	0.10	0.5	0.6	0.9	1.4	1.7	1.6	1.0
France	1980-1982	29	8.25	0.2	0.04	0.07	0.5	0.4	0.5	1.2	1.7	1.9	1.6
Finland	1980-1983	30	8.55	0.1	0.05	0.08	0.4	0.5	0.7	1.3	2.0	2.1	1.3
USSR	1985/86	31	9	..	..	..	..	..	..	..	..	..	..
Means, 30 countries[b]			6.60[b]	0.2	0.04	0.08	0.4	0.3	0.4	0.9	1.4	1.6	1.2

Sources: Except for the USSR, based on a set of life tables calculated by the Population Division of the United Nations Secretariat for the present study (see table 2 sources). The contributions of age groups to the sex differential in life expectancy at birth have been obtained by applying the formulae given in note 2 of the text to the set of life tables.

NOTE: Because of rounding, the sum of the contributions for the 10 age groups for a country may differ slightly from the sex differential in life expectancy in column (1).

[a] Because of the small size of the contributions, values are shown to two decimal places.

[b] Excluding USSR. Mean sex differential in life expectancy including USSR is 6.68 years.

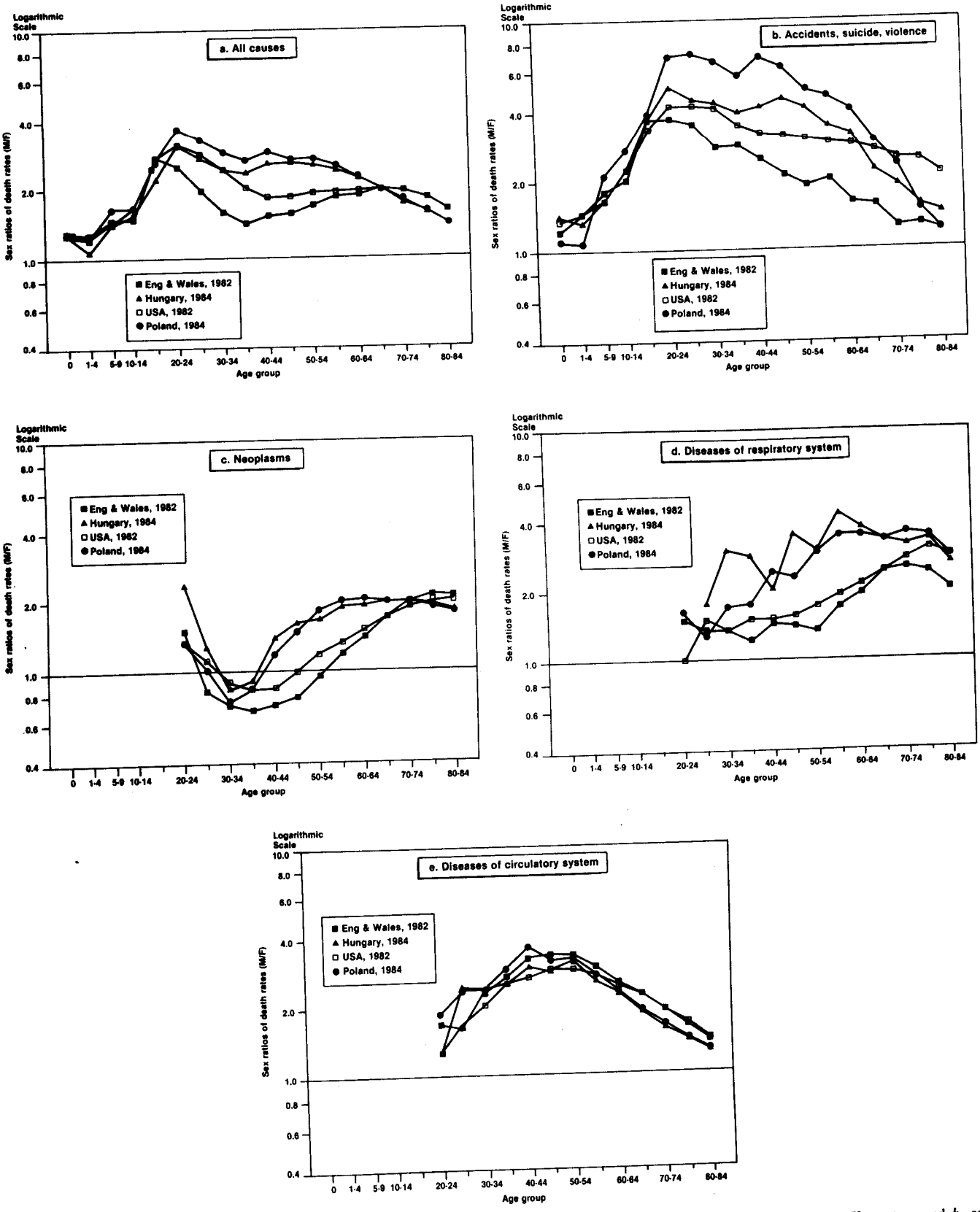
for the observed variations among countries in the size of the sex differential in life expectancy at birth. At ages 15-24, however, countries in the lower half of the table tend to have higher contributions (0.4, 0.5 and 0.6 year) than countries in the upper part of the table (0.3 year); this age group therefore accounts for some of the country variations in the size of the sex differential in life expectancy, but only for a few tenths of a year.

Differences between male and female mortality at ages 25-34 contributed from 0.2 year to 0.6 year to the sex differential in life expectancy. The average contribution from this age group (0.35 year) is very close to that of ages 15-24 (0.41 year). While sex ratios of death rates are lower at ages 25-34 than at 15-24 (the average ratios are 2.3 and 2.7, respectively), differences between male and female death rates are larger, because the death rates themselves are larger. At ages 25-34, contributions from the countries towards the bottom of table 4 are somewhat larger than those in the upper part of the table, accounting for a few tenths of a year of the variation among countries in the size of the sex differential in life expectancy.

At ages 35-44, contributions to the sex differential in life expectancy ranged from 0.2 year in England and Wales (United Kingdom), the Netherlands and New Zealand, to 0.9 year in Hungary and Poland, and averaged 0.44 year. The countries in the lower part of table 4 generally had contributions that were 0.2 or 0.3 year higher than the group average. Contrary to expectation, several countries in the upper part of the table (namely, Bulgaria, Romania and Yugoslavia) also had above average contributions of 0.6 or 0.7 year.

Age group 45-54 is the first in which contributions of a year or more are observed. Contributions range from 0.5 year in Denmark, Greece and New Zealand, to 1.4 years in Hungary and Poland, with an average of 0.9

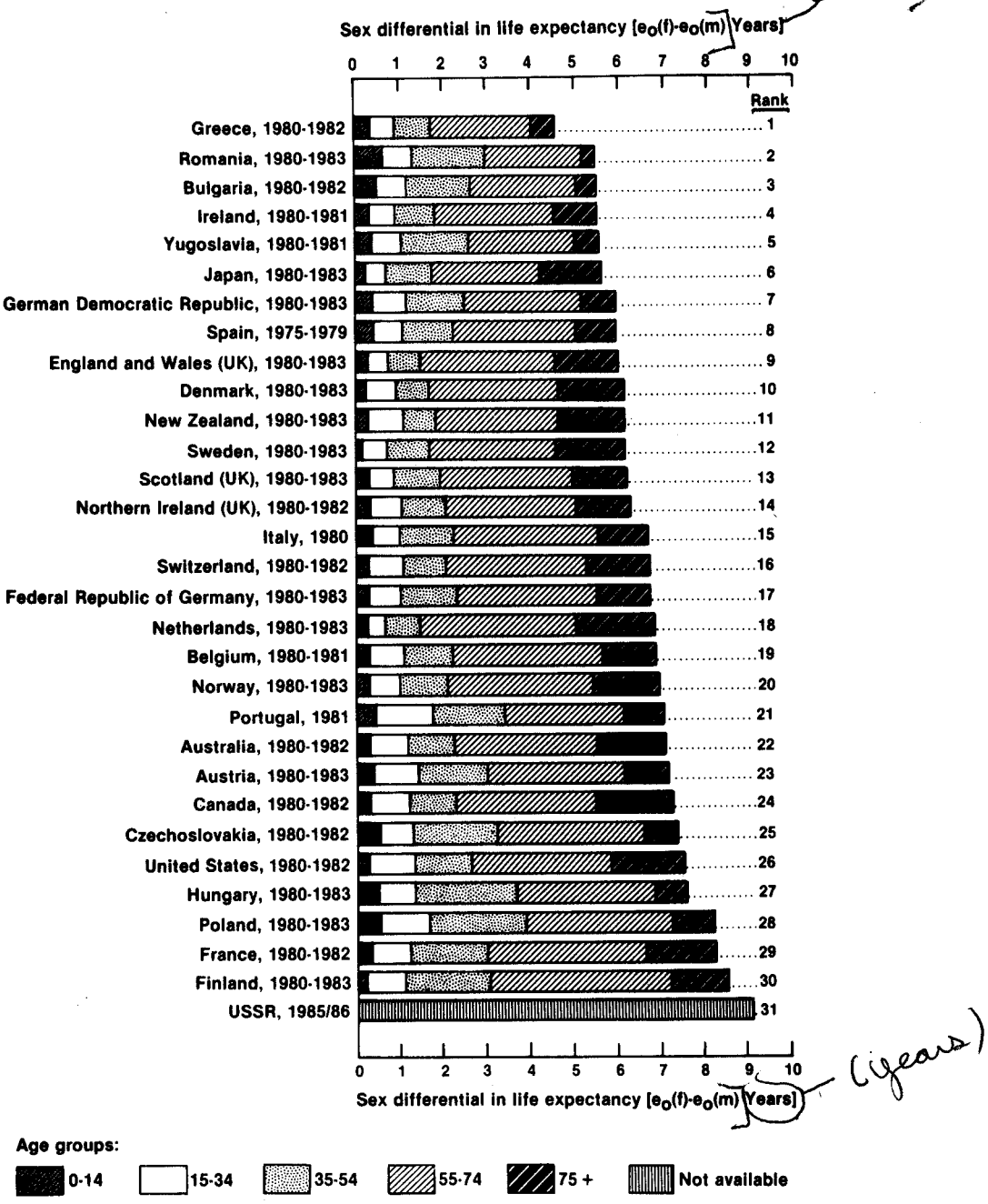
Figure III. Comparison of sex ratios of death rates by age group and cause of death in selected developed countries with "Western" and "Eastern" patterns of sex ratios,[a] early 1980s



Source: World Health Organization data bank. For the composition of the cause-of-death groups, see table 7, note a.

[a] For a description of the patterns, see figure II, parts *a* and *b*, and the related discussion.

Figure IV. Contribution of age groups to the sex differential in life expectancy at birth, developed countries ranked according to size of sex differential, early 1980s or latest available period



Source: Table 4.

year. Most of the countries with the largest contributions at ages 45-54 are those with the largest sex differential in life expectancy at birth (Czechoslovakia, Finland, France, Hungary and Poland).

The average contribution to the sex differential in life expectancy at ages 55-64 was 1.4 years, with a range from 1.0 year in Japan to 2.0 years in Finland. The countries with the largest contributions at ages 55-64 (1.7 to 2.0 years) were the same ones mentioned for ages 45-54.

Of the 10 age groups, the average contribution was highest at ages 65-74 (1.6 years). Contributions from this age group ranged from 1.0 year (Romania) to 2.1 years (Finland and the Netherlands).

The oldest age group, 75 and over, shows the most variability in the size of contributions, which range from 0.3 year in Romania to 1.8 years in Canada and the Netherlands, with an average of 1.2 years. Some of this variability may be due to differences among countries in the age structure of the old-age population.

TABLE 5. PERCENTAGE CONTRIBUTION OF 10 AGE GROUPS TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, DEVELOPED COUNTRIES RANKED BY SIZE OF THE SEX DIFFERENTIAL, EARLY 1980S OR LATEST AVAILABLE PERIOD

Country	Period	Rank	Contribution of age group to the sex differential in life expectancy (percentage)										
			All ages	0	1-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75 and over
Greece	1980-1982	1	100.0	5.7	0.8	1.3	6.9	5.5	6.3	11.9	24.4	25.2	12.0
Romania	1980-1983	2	100.0	7.6	1.0	3.2	5.6	6.8	12.2	18.2	21.8	17.9	5.7
Bulgaria	1980-1982	3	100.0	6.6	0.8	1.8	5.8	6.8	10.0	16.4	23.3	19.9	8.5
Ireland	1980-1981	4	100.0	2.6	1.0	2.0	6.2	4.4	5.3	11.6	22.7	26.5	17.7
Yugoslavia	1980-1981	5	100.0	4.7	0.5	1.5	5.3	7.0	10.0	17.9	23.0	20.1	10.1
Japan	1980-1983	6	100.0	2.0	0.8	1.3	4.8	3.5	5.8	13.1	18.2	25.3	25.4
German Democratic Republic	1980-1983	7	100.0	4.7	0.6	1.3	6.8	6.1	7.4	14.6	20.8	23.9	13.6
Spain	1975-1979	8	100.0	4.9	0.8	1.5	5.7	5.1	6.9	13.3	21.8	25.6	14.6
England and Wales (UK)	1980-1983	9	100.0	3.4	0.4	0.9	4.4	2.9	3.4	9.3	21.5	29.6	24.0
Denmark	1980-1983	10	100.0	2.6	0.5	0.7	5.3	5.3	4.4	8.6	20.3	28.0	24.3
New Zealand	1980-1983	11	100.0	3.0	0.7	1.1	7.8	5.2	3.7	8.1	20.4	25.6	24.4
Sweden	1980-1983	12	100.0	1.2	0.3	0.8	4.2	4.9	5.7	10.6	19.8	27.0	25.6
Scotland (UK)	1980-1983	13	100.0	3.3	0.9	1.1	5.1	4.0	5.0	11.8	22.8	25.9	20.2
Northern Ireland (UK)	1980-1982	14	100.0	3.6	0.9	0.7	6.5	5.0	4.5	11.5	21.2	26.4	19.6
Italy	1980	15	100.0	4.5	0.2	1.1	5.6	3.6	5.3	13.7	23.9	25.4	16.9
Switzerland	1980-1982	16	100.0	2.3	0.8	1.1	7.2	4.7	4.8	9.7	20.6	27.1	21.7
Germany, Federal Republic of	1980-1983	17	100.0	2.9	0.4	1.1	6.2	4.5	6.5	12.9	20.8	26.5	18.2
Netherlands	1980-1983	18	100.0	2.3	0.3	1.0	3.5	2.5	3.1	8.7	21.2	31.0	26.3
Belgium	1980-1981	19	100.0	4.0	−0.0	0.7	6.7	4.9	5.2	11.1	22.4	27.3	17.8
Norway	1980-1983	20	100.0	2.1	0.7	1.3	6.1	4.3	4.6	11.6	20.8	26.6	21.8
Portugal	1981	21	100.0	3.6	1.4	1.5	11.4	7.3	8.7	14.4	19.3	19.2	13.2
Australia	1980-1982	22	100.0	2.6	0.6	1.3	7.3	5.0	4.6	10.5	19.5	26.2	22.4
Austria	1980-1983	23	100.0	4.2	0.5	0.8	8.5	6.0	7.5	14.9	20.7	22.9	14.0
Canada	1980-1982	24	100.0	2.6	0.6	1.2	7.3	5.1	4.7	10.4	19.3	24.5	24.4
Czechoslovakia	1980-1982	25	100.0	5.5	0.6	1.2	4.8	5.6	9.2	16.8	24.0	21.4	10.9
United States	1980-1982	26	100.0	2.3	0.6	1.0	7.3	6.8	6.3	11.1	18.8	23.2	22.6
Hungary	1980-1983	27	100.0	4.8	0.7	1.0	5.1	6.4	11.7	18.9	22.5	18.9	10.0
Poland	1980-1983	28	100.0	5.0	0.6	1.3	5.9	7.5	10.6	16.6	20.7	20.0	12.1
France	1980-1982	29	100.0	2.7	0.5	0.9	6.1	5.1	6.4	14.7	20.8	23.3	19.7
Finland	1980-1983	30	100.0	1.1	0.6	0.9	4.4	5.8	7.7	15.3	24.0	24.3	15.7
Means, 30 countries				3.6	0.6	1.2	6.1	5.2	6.6	12.9	21.4	24.5	17.8

Source: Based on the values calculated for table 4 before they were rounded to one decimal place.

NOTE: Because of rounding, the sum of the percentages for the 10 age groups for a country may not in all cases equal 100.0 per cent.

The percentage contributions of the 10 age groups to the sex differential in life expectancy are shown in table 5. Based on the average values for the 30 countries, age 0 contributed about 4 per cent of the total and ages 1-4 only about half a percentage point. Between ages 1-4 and 65-74, the percentage contributions tend to increase with advancing age, reaching 21.4 per cent at ages 55-64, and 24.5 per cent at ages 65-74. Ages 75 and over contributed an average of 17.8 per cent.

D. *Contribution of causes of death to the sex differential in life expectancy*

One indicator of the quality of cause-of-death statistics is the percentage of deaths assigned to senility and ill-defined causes. Large variations among countries in this percentage may impair international comparability. A low proportion of deaths with non-specific diagnoses, however, is not necessarily evidence of accurate data, as countries may distribute such deaths by imputing procedures among the known causes (Hansluwka, 1978, p. 166).

The percentage of deaths classified as being due to senility and ill-defined conditions in the early 1980s averaged 3.0 per cent for 25 of the 26 countries included in tables 7 through 10 for which such data were available. The countries with the largest percentages were Portugal (12 per cent), Yugoslavia (9 per cent) and Poland and France (7 per cent each). Less than one per cent of all deaths were so classified in Austria, Australia, Czechoslovakia, Finland, Hungary, Romania, Sweden, England and Wales and Scotland. Most of such deaths occur in the oldest age groups, where multiple morbid conditions make selection of an underlying cause difficult.

Male mortality is higher than female mortality for most causes of death in developed countries today. The major exceptions are, of course, conditions that affect females exclusively (namely, diseases of the female breast and reproductive system). One cause of death for which female mortality is higher in some countries is congenital anomalies of the central nervous system, an infrequent cause of early infant mortality (Waldron, 1986b). Earlier in the century, females had higher mortality than males from cerebro-vascular disease in many developed countries (Moriyama, Krueger and Stamler, 1971, pp. 216 and 224), but that disadvantage has all but disappeared. Higher female mortality from diabetes mellitus is still found at the older ages in many developed

countries, but the gap with males has also been narrowing.

Data on sex differentials in mortality in the early 1980s by cause of death are presented in tables 6 to 11 for 25 to 28 countries. Table 6 shows sex ratios of death rates for selected causes based on age-standardized death rates, a measure that summarizes mortality at all ages in a population and eliminates the effects of differences in age structure among the populations being compared. The most striking data in table 6 are the high sex ratios for cancer of the respiratory system, which is the leading cause of cancer mortality among males in most developed countries. On average, males were about six times more likely to die from this cause than females. The highest ratios were in Belgium (13.2), Finland (11.5), the Netherlands (11.1) and France (10.8).

Cigarette smoking is the major cause of sex differentials in mortality from lung cancer (Waldron, 1986a). Although not shown separately in table 6, sex ratios of death rates for other cancer sites affected by tobacco use (e.g., oral cavity and larynx) are also high (United States, 1982, pp. 63-79). However, cancers at those sites are far less important numerically than lung cancer, hence have a smaller effect on overall sex differentials in mortality.

Sex ratios of death rates for stomach cancer averaged about 2 for the countries in table 6. The ratios are much less variable than for lung cancer, and range from 1.6 in New Zealand to 2.6 in Poland. In contrast to other developed countries, in Japan and Portugal stomach cancer is the leading cause of cancer deaths for both sexes.

The average sex ratio of death rates for all neoplasms combined was relatively low (1.7) compared to the ratio for respiratory cancers. The low ratio reflects the fact that several cancer sites common to both sexes display lower than average sex ratios (the most important of these numerically is colon cancer), and partly because cancer mortality from the exclusively female sites is considerably higher than from the exclusively male sites.

Of the causes shown in table 6, the highest average sex ratios of death rates, after cancer of the respiratory system, were for chronic respiratory diseases (bronchitis, emphysema and asthma) and for motor vehicle traffic accidents, both groups having ratios of 3.1. Not coincidentally, the countries with the highest ratios for chronic respiratory diseases are the same as for respiratory cancer: Finland (ratio of 7.0), Netherlands (5.3), Belgium and France (4.3 each).

For the group of external causes (accidents, suicide and violence), the average sex ratio was 2.5. The ratios for motor vehicle traffic accidents averaged 3.1, with a range from 2.3 in New Zealand to 4.5 in Poland and 4.7 in Portugal. The mean sex ratio for suicide deaths was 2.8, with the highest ratios observed for Finland (4.1) and Poland (4.9). Chronic liver disease and cirrhosis displayed a mean ratio of 2.5, with a range from 1.4 (in England and Wales, Scotland and Ireland) to 3.4 in Czechoslovakia and 3.6 in Switzerland.

Diseases of the circulatory system account for about half of all deaths in developed countries. The most important component numerically, ischaemic heart disease, displayed a mean ratio of 2.2 and a range from 1.4 in Romania to 3.1 in Poland. The ratio for cerebro-vascular disease (1.2) is the lowest mean ratio of the causes shown in table 6. In Greece and Northern Ireland, the age-standardized death rates for cerebro-vascular disease were marginally higher for females, but the sex differentials were too small to affect the sex ratios of death rates for that cause of death, which were 1.0 in those countries.

The average sex ratio for mortality from the infectious and parasitic diseases was 1.8, with a range from 1.2 in Northern Ireland to 2.8 in Poland and 2.9 in Hungary. The infectious diseases have a negligible impact on overall patterns of sex differentials because they currently account for such a small proportion of deaths in developed countries.

Tables 7 through 11, and figure V present the results of a decomposition of sex differentials in life expectancy at birth by causes of death for the early 1980s.[3] Diseases of the circulatory system were the largest contributors to the sex differential in life expectancy in all but three countries (France, Japan and Romania), accounting for an average of 2.7 years of the total differential of 6.8 years (table 7). The exceptions were France, Japan and Romania. The smallest absolute contributions from the circulatory diseases were in Romania (1.6 years), Japan and Yugoslavia (1.7 years in each), while the largest were in countries of Northern Europe: Finland, 3.9 years; Norway, 3.4 years; and Sweden, 3.3 years. In relative terms, circulatory diseases accounted for an average of about 40 per cent of the sex differential in life expectancy (table 8). The smallest percentage contributions (between about 25 and 30 per cent) were in France, Japan, Portugal, Romania and Yugoslavia, while the largest (about 50 per cent) were in the Northern European countries and New Zealand.

Accidents, suicide and violence contributed an average of 1.3 years to the sex differential in life expectancy, with a range from 0.6 year in England and Wales and the Netherlands, to 1.9 years in Poland. Large contributions of 1.7 or 1.8 years were also found in Austria, Finland, Hungary, Portugal and the United States. These causes were the second largest contributor to the sex differential in about half the countries. In relative terms, the contributions averaged 19 per cent, with a range from about 9 per cent (Netherlands) to over 25 per cent (Austria, Portugal, Romania).

Neoplasms accounted for 1.2 years, on average, of the sex differential in life expectancy, only slightly less than the external causes. France had an unusual pattern in which neoplasms were the largest contributor to the sex differential (2.3 years), contributing even more than diseases of the circulatory system (2.0 years). In Japan, the contribution from neoplasms and diseases of the circulatory system were about the same (1.7 years). Other countries with large contributions from neoplasms were the Netherlands (1.9 years) and Belgium, Italy and Switzerland (1.7 years each). The smallest contributions were in Bulgaria, Romania and Sweden (0.7 year each). The relative contribution of neoplasms averaged 18 per

TABLE 6. SEX RATIOS OF AGE-STANDARDIZED DEATH RATES FOR SELECTED CAUSES OF DEATH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	All causes	Infectious and parasitic diseases	Malignant neoplasms			Diseases of circulatory system			Diseases of respiratory system		Chronic liver disease and cirrhosis	Accidents, suicide, violence		
				All	Stomach	Trachea, bronchus, lung	All	Ischaemic heart disease	Cerebro-vascular disease	All	Chronic bronchitis, emphysema, asthma		All	Motor vehicle traffic accidents	Suicide
Northern America															
Canada	1982	1.7	1.5	1.6	2.2	3.8	1.7	2.0	1.1	2.3	3.0	2.3	2.5	2.5	3.4
United States	1982	1.7	1.5	1.6	2.2	3.1	1.7	2.0	1.1	2.3	2.5	2.2	2.9	2.8	3.4
East Asia															
Japan	1984	1.6	2.2	1.9	2.2	3.6	1.4	1.7	1.4	2.2	2.6	3.0	2.6	3.5	2.2
Europe															
Eastern Europe															
Bulgaria	1983	1.5	2.1	1.6	1.9	6.3	1.3	1.6	1.2	1.8	2.3	3.0	3.5	3.3	2.9
Czechoslovakia	1983	1.7	1.7	1.9	2.2	9.7	1.5	1.8	1.2	2.1	3.3	3.4	2.2	3.0	3.6
Hungary	1984	1.7	2.9	1.8	2.4	5.9	1.5	2.0	1.3	2.7	2.9	2.9	2.4	3.8	3.0
Poland	1984	1.7	2.8	1.8	2.6	7.8	1.6	3.1	1.2	2.8	3.9	2.5	3.4	4.5	4.9
Romania	1983	1.3	2.2	1.5	2.3	5.6	1.2	1.4	1.1	1.6	1.5	1.9	3.1	..	..
Northern Europe															
Denmark	1982	1.6	1.4	1.4	1.9	3.5	1.7	2.1	1.3	2.0	2.7	1.7	1.8	2.6	1.8
Finland	1983	1.9	1.8	1.8	1.8	11.5	1.8	2.5	1.2	2.7	7.0	2.9	3.3	2.3	4.1
Ireland	1981	1.6	1.6	1.4	1.7	3.1	1.6	2.2	1.0	1.8	2.2	1.4	2.4	3.2	2.2
Norway	1983	1.7	1.5	1.5	2.0	4.2	1.8	2.5	1.2	1.8	2.9	2.1	2.4	2.6	2.6
Sweden	1982	1.7	1.4	1.3	2.0	3.1	1.8	2.2	1.1	1.8	2.4	3.0	2.4	2.5	2.5
United Kingdom															
England and Wales	1982	1.6	1.6	1.5	2.2	3.8	1.7	2.3	1.1	1.9	3.6	1.4	1.9	2.7	2.1
Northern Ireland	1981	1.6	1.2	1.4	1.7	3.7	1.7	2.2	1.0	1.8	3.1	2.2	2.5	..	..
Scotland	1984	1.6	1.7	1.5	1.8	3.1	1.7	2.1	1.2	1.9	2.7	1.4	2.0	2.9	2.6
Southern Europe															
Greece	1982	1.4	2.0	1.8	1.7	7.2	1.2	2.6	1.0	1.5	2.0	2.9	2.2	3.2	2.6
Italy	1980	1.7	2.1	1.9	2.1	8.9	1.5	2.0	1.3	2.4	3.4	3.0	2.2	3.8	2.4
Portugal	1982	1.6	2.3	1.6	2.0	5.0	1.4	1.9	1.3	2.0	2.4	3.1	3.3	4.7	2.8
Yugoslavia	1982	1.5	1.5	1.7	2.1	6.0	1.3	2.1	1.2	1.7	2.4	3.2	2.9	3.8	2.4
Western Europe															
Austria	1982	1.7	2.6	1.6	2.0	6.2	1.5	2.3	1.3	2.2	2.8	3.2	2.5	3.5	2.9
Belgium	1979	1.7	1.9	1.9	2.0	13.2	1.6	2.4	1.2	3.1	4.3	2.1	1.8	3.0	2.0
France	1981	1.9	1.8	2.2	2.3	10.8	1.6	2.3	1.4	2.4	3.0	2.7	2.2	2.9	2.8
Germany, Federal Republic of	1984	1.7	1.9	1.6	1.9	7.4	1.6	2.3	1.3	2.6	3.7	2.7	2.1	2.7	2.4
Netherlands	1983	1.8	1.3	1.9	2.4	11.1	1.8	2.4	1.2	2.7	5.3	2.1	1.8	2.7	1.6
Switzerland	1984	1.7	1.4	1.7	2.0	8.2	1.7	2.6	1.2	2.7	4.3	3.6	2.3	3.3	2.6
Oceania															
Australia-New Zealand															
Australia	1983	1.7	1.7	1.7	2.2	4.4	1.6	2.0	1.1	2.7	2.7	2.6	2.6	2.6	3.1
New Zealand	1983	1.6	1.4	1.4	1.6	3.9	1.7	2.1	1.1	2.0	2.6	2.0	2.2	2.3	2.4
Means, 28 countries[a]		1.6	1.8	1.7	2.1	6.2	1.6	2.2	1.2	2.2	3.1	2.5	2.5	3.1	2.8

Sources: Ratios calculated from age-standardized death rates in World Health Organization, *World Health Statistics Annual 1984* (Geneva, 1984) and *World Health Statistics Annual 1985* (Geneva, 1985), table 16.

NOTE: Deaths are classified according to the Ninth Revision of the International Classification of Diseases (ICD) in all countries but Denmark, Finland, Norway, Sweden and Switzerland, which use the Eighth Revision. The codes of the ICD for the causes of death included in this table are given in footnotes to tables 7, 9, 10 and 11, with these exceptions:

	Eighth Revision (Intermediate List of 150 Causes)	*Ninth Revision (Basic Tabulation List)*
Malignant neoplasms of stomach	A47	091
Chronic bronchitis, emphysema, asthma	A93	323
Chronic liver disease and cirrhosis	A102	347

[a] For motor vehicle traffic accidents and suicide, means are for 26 countries.

TABLE 7. CONTRIBUTION OF BROAD GROUPS OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	Revision of the ICD	Contribution of causes of death[a] to the sex differential in life expectancy (years)							
			All causes (1)	Infectious and parasitic diseases (2)	Neoplasms (3)	Diseases of circulatory system (4)	Diseases of respiratory system (5)	Accidents, suicide, violence (6)	Ill-defined conditions (7)	All other causes (residual) (8)
Northern America										
Canada	1983	9th	7.2	0.0	1.3	3.1	0.7	1.4	0.1	0.6
United States	1982	9th	7.5	0.1	1.2	3.2	0.6	1.7	0.2	0.6
East Asia										
Japan	1984	9th	5.9	0.1	1.7	1.7	0.7	1.1	0.0	0.6
Europe										
Eastern Europe										
Bulgaria	1982	9th	5.5	0.1	0.7	2.0	0.6	1.3	0.1	0.7
Czechoslovakia	1982	9th	7.4	0.0	1.4	2.9	0.6	1.4	0.0	1.0
German Democratic Republic	1984	9th	5.8	0.0	1.0	2.2	0.6	0.7	0.0	1.3
Hungary	1984	9th	8.2	0.1	1.4	3.1	0.6	1.8	0.0	1.2
Poland	1984	9th	8.2	0.2	1.4	3.0	0.6	1.9	0.5	0.7
Romania	1983	9th	5.6	0.1	0.7	1.6	0.8	1.6	0.0	0.8
Northern Europe										
Denmark	1984	8th	6.0	0.0	0.8	2.8	0.5	0.9	0.3	0.7
Finland	1983	8th	8.3	0.1	1.3	3.9	0.8	1.8	0.0	0.4
Norway	1984	8th	6.8	0.0	0.9	3.4	0.5	1.2	0.3	0.4
Sweden	1984	8th	6.2	0.0	0.7	3.3	0.4	1.1	0.1	0.6
United Kingdom										
England and Wales	1982	9th	5.9	0.0	1.0	3.0	0.9	0.6	0.0	0.3
Scotland	1984	9th	6.0	0.0	0.9	3.1	0.7	0.8	0.1	0.4
Southern Europe										
Italy	1980	9th	6.7	0.1	1.7	2.1	0.6	1.0	0.1	1.1
Portugal	1984	9th	7.0	0.1	0.9	1.8	0.7	1.8	0.6	1.1
Yugoslavia	1982	9th	5.9	0.1	1.0	1.7	0.4	1.4	0.3	1.1
Western Europe										
Austria	1984	9th	7.2	0.1	1.2	2.7	0.4	1.8	0.0	1.0
Belgium	1982	9th	6.8	0.1	1.7	2.3	0.8	1.2	0.2	0.4
France	1983	9th	8.2	0.1	2.3	2.0	0.6	1.6	0.5	1.1
Germany, Federal Republic of	1984	9th	6.7	0.1	1.3	2.8	0.6	1.0	0.2	0.8
Netherlands	1984	9th	6.9	0.0	1.9	2.9	0.7	0.6	0.3	0.5
Switzerland	1984	8th	6.9	0.0	1.7	2.6	0.5	1.4	0.1	0.6
Oceania										
Australia-New Zealand										
Australia	1983	9th	6.8	0.0	1.3	2.9	0.8	1.2	0.1	0.5
New Zealand	1983	9th	6.2	0.0	0.8	3.0	0.8	1.2	0.1	0.3
Means, 26 countries			6.8	0.1	1.2	2.7	0.6	1.3	0.2	0.7

Source: Based on a set of life tables calculated by the Population Division of the United Nations Secretariat from registered deaths and population estimates in the World Health Organization data bank. The contributions of causes of death to the sex differential in life expectancy have been obtained by applying the formulae in notes 2 and 3 of the text.

NOTE: Because of rounding, the sum of the contributions shown for each cause-of-death group for a country may differ slightly from the total for all causes in column (1).

[a] The codes of the International Classification of Diseases for the causes of death shown are as follows:

	Eighth Revision (Intermediate List of 150 Causes)	Ninth Revision (Basic Tabulation List)
Infectious and parasitic diseases	A1-A44	01-07
Neoplasms (malignant, benign, unspecified)	A45-A61	08-17
Diseases of circulatory system	A80-A88	25-30
Diseases of respiratory system	A89-A96	31-32
Accidents, suicide, violence	AE138-AE150	E47-E56
Ill-defined conditions	A136-A137	46
All other (residual)	A62-A79, A97-A135	18-24, 34-45

cent, with a range from about 12 per cent in Romania and Sweden to 28 or 29 per cent in France, Japan and the Netherlands.

As pointed out by Preston, Keyfitz and Schoen (1972, p. 4), the International Classification of Diseases, in its successive revisions, has shifted gradually from anatomic to aetiological criteria in classifying causes of death, but elements of an anatomic classification persist. This is clearly the case with the category "Diseases of the respiratory system", which includes chronic diseases (chronic bronchitis, emphysema and asthma) and acute infectious diseases (influenza and viral and bacterial pneumonia, among others). Diseases of the respiratory system accounted for 0.6 year, on average, of the sex differential in life expectancy, with a rather narrow country range from 0.4 to 0.9 year. These diseases accounted for about 10 per cent of the total sex differential, with a range from about 6 per cent in Austria and Yugoslavia to

TABLE 8. PERCENTAGE CONTRIBUTION OF BROAD GROUPS OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	Revision of the ICD	Contribution of causes of death[a] to the sex differential in life expectancy (percentage)							
			All causes (1)	Infectious and parasitic diseases (2)	Neoplasms (3)	Diseases of circulatory system (4)	Diseases of respiratory system (5)	Accidents, suicide, violence (6)	Ill-defined conditions (7)	All other causes (residual) (8)
Northern America										
Canada	1983	9th	100.0	0.6	18.0	42.7	9.9	19.4	1.5	8.0
United States	1982	9th	100.0	0.7	16.1	42.5	8.3	22.1	2.1	8.4
East Asia										
Japan	1984	9th	100.0	2.3	28.7	28.3	12.1	18.8	0.4	9.4
Europe										
Eastern Europe										
Bulgaria	1982	9th	100.0	1.2	13.2	35.8	11.3	24.3	1.3	12.9
Czechoslovakia	1982	9th	100.0	0.6	19.4	39.3	8.5	18.6	0.6	13.0
German Democratic Republic	1984	9th	100.0	0.6	16.7	38.6	10.6	11.3	0.0	22.1
Hungary	1984	9th	100.0	1.5	16.8	37.7	6.9	22.5	0.2	14.4
Poland	1984	9th	100.0	2.0	16.6	36.3	7.3	23.3	6.0	8.6
Romania	1983	9th	100.0	2.6	12.3	28.5	13.8	27.7	0.0	15.0
Northern Europe										
Denmark	1984	8th	100.0	0.3	13.5	47.5	8.0	15.3	4.4	11.0
Finland	1983	8th	100.0	0.7	15.6	47.7	9.4	21.7	0.0	4.9
Norway	1984	8th	100.0	0.6	13.6	50.7	7.1	18.0	3.7	6.3
Sweden	1984	8th	100.0	0.3	11.6	52.1	6.6	18.3	0.7	10.3
United Kingdom										
England and Wales	1982	9th	100.0	0.5	16.2	51.1	15.0	10.6	0.8	5.7
Scotland	1984	9th	100.0	0.4	15.1	51.7	11.3	13.9	1.0	6.5
Southern Europe										
Italy	1980	9th	100.0	1.0	24.8	32.0	9.6	15.2	1.0	16.4
Portugal	1984	9th	100.0	2.0	13.2	25.6	9.3	25.6	8.3	16.1
Yugoslavia	1982	9th	100.0	1.0	16.9	28.8	6.1	23.5	5.8	18.0
Western Europe										
Austria	1984	9th	100.0	1.0	16.2	36.8	5.6	25.3	0.7	14.3
Belgium	1982	9th	100.0	0.8	24.9	34.5	11.8	18.4	3.5	6.1
France	1983	9th	100.0	1.0	28.7	24.5	6.9	19.4	6.3	13.4
Germany, Federal Republic of	1984	9th	100.0	1.0	19.1	41.8	8.6	14.8	3.0	11.6
Netherlands	1984	9th	100.0	0.3	28.0	42.1	9.6	9.1	4.0	6.8
Switzerland	1984	8th	100.0	0.5	24.5	36.9	7.4	20.7	1.3	8.7
Oceania										
Australia-New Zealand										
Australia	1983	9th	100.0	0.5	18.7	42.6	11.2	18.0	1.2	7.9
New Zealand	1983	9th	100.0	0.3	12.8	48.1	12.9	18.8	2.1	5.0
Means, 26 countries			100.0	0.9	18.1	39.4	9.5	19.0	2.3	10.8

Source: Based on the values calculated for table 7 before they were rounded to one decimal place.

NOTE: Because of rounding, the sum of the percentages for the cause-of-death groups for a country may not in all cases equal 100.0 per cent.

[a] For the codes of the International Classification of Diseases, see table 7, note a.

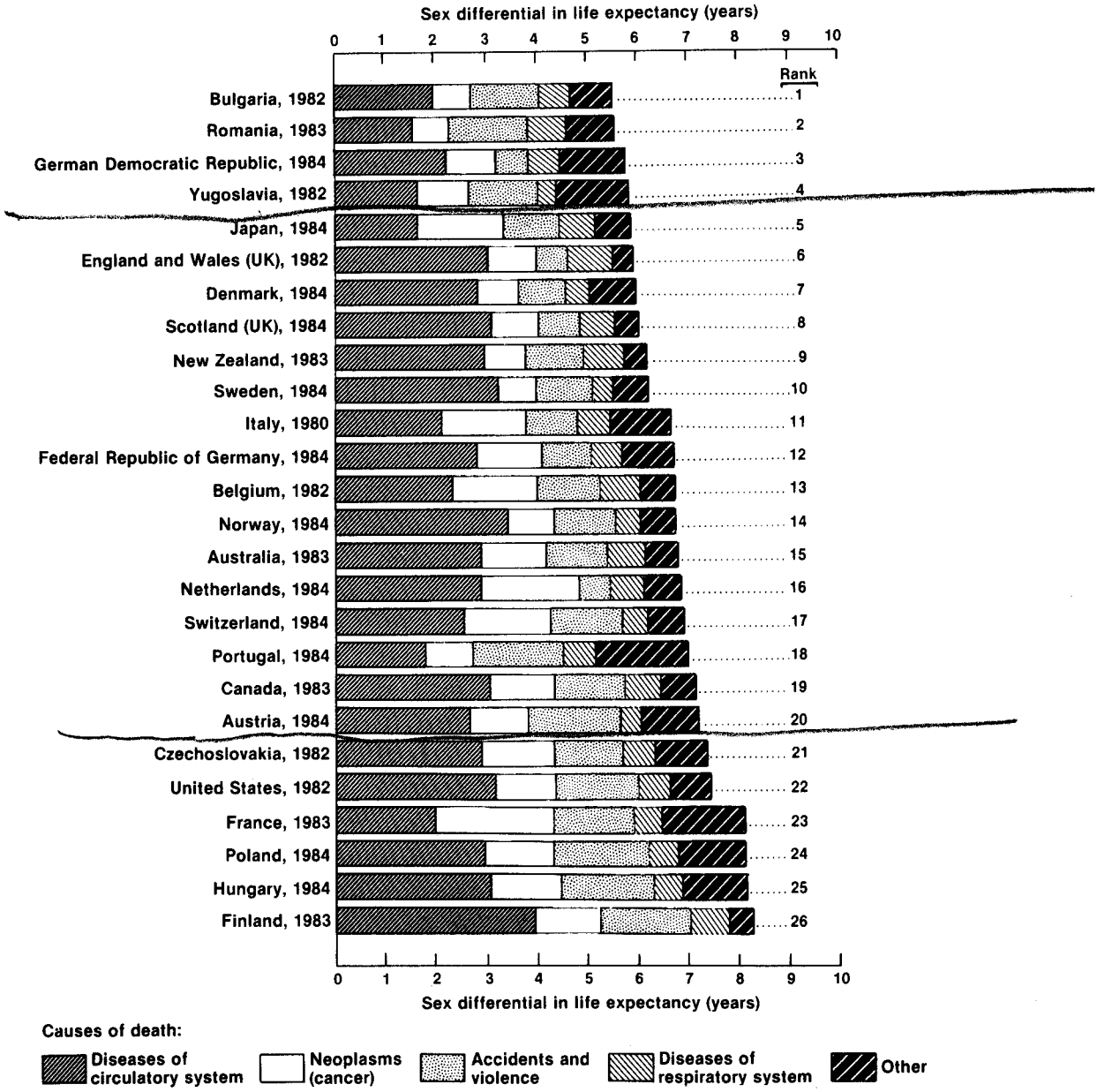
15 per cent in England and Wales. Of the 0.6 year average contribution from respiratory diseases, 0.2 year is attributable to pneumonia and influenza and 0.4 year to chronic bronchitis, emphysema and asthma.

The infectious and parasitic diseases account for less than 1 per cent of all deaths in developed countries. The average contribution to the sex differential from these causes was under 0.1 year.

The category of ill-defined conditions contributed an average of 0.2 year and 2.3 per cent to the sex differential in life expectancy. The largest contributions were in Portugal (0.6 year), France and Poland (0.5 year each). To the extent that the deaths included here would have been classified elsewhere had all the necessary information on cause of death been available, the contributions to the sex differential for some causes of death may be slightly understated.

Tables 9 to 11 present more detailed data by cause for three of the major cause-of-death groups. Ischaemic heart disease, the leading cause of death in most developed countries, contributed an average of 1.8 years (27 per cent) to the sex differential in life expectancy (table 9). The contributions were greatest in countries of Northern Europe: the largest absolute contribution was in Finland (3.3 years) and the largest relative contributions (44 or 45 per cent) were in Sweden, England and Wales and Scotland. In most of the countries (17 out of 26), ischaemic heart disease contributed more to the sex

Figure V. Contribution of causes of death to the sex differential in life expectancy at birth, developed countries ranked according to size of sex differential, latest available year



Source: Table 7.

differential in life expectancy than all neoplasms combined. Some major exceptions were Japan and France, where ischaemic heart disease accounted for only 7 per cent and 11 per cent, respectively, of the sex differential. Relatively low contributions (between 12 and 15 per cent) were also evident for Portugal, Romania and Yugoslavia.

The second major component of the circulatory diseases, cerebro-vascular disease, contributed an average of only 0.3 year (4.3 per cent) to the sex differential. As mentioned earlier, sex ratios of death rates are relatively low for this cause (as shown in table 6, the average sex ratio for all ages in the early 1980s was 1.2 for cerebro-vascular disease, compared with 2.2 for ischaemic heart disease). Above average contributions from cerebro-vascular disease are seen for Japan (0.7 year and 12 per cent) and Portugal (0.6 year and 9 per cent).

Table 10 shows the contributions to the sex differential of malignant neoplasms of selected sites: cancer of the trachea, bronchus and lung (to be referred to as lung cancer), the leading sites for males in most developed countries; breast cancer, similarly the leading site for

TABLE 9. CONTRIBUTION OF MAJOR DISEASES OF THE CIRCULATORY SYSTEM TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	Revision of the ICD	Contribution of diseases of the circulatory system[a]					
			All circulatory diseases		Major diseases			
					Ischaemic heart disease[b]		Cerebro-vascular disease	
			Years	Per cent	Years	Per cent	Years	Per cent
Northern America								
Canada	1983	9th	3.1	42.7	2.4	33.9	0.1	2.0
United States	1982	9th	3.2	42.5	2.3	31.3	0.1	1.7
East Asia								
Japan	1984	9th	1.7	28.3	0.4	7.0	0.7	11.8
Europe								
Eastern Europe								
Bulgaria	1982	9th	2.0	35.8	1.1	19.8	0.4	7.2
Czechoslovakia	1982	9th	2.9	39.3	1.9	25.6	0.5	6.4
German Democratic Republic	1984	9th	2.2	38.6	1.3	21.8	0.2	3.1
Hungary	1984	9th	3.1	37.7	1.9	22.8	0.6	7.4
Poland	1984	9th	3.0	36.3	1.4	17.6	0.2	2.2
Romania	1983	9th	1.6	28.5	0.8	14.7	0.2	4.2
Northern Europe								
Denmark	1984	8th	2.8	47.5	2.4	39.3	0.2	2.8
Finland	1983	8th	3.9	47.7	3.3	39.4	0.3	3.3
Norway	1984	8th	3.4	50.7	2.7	40.3	0.3	4.3
Sweden	1984	8th	3.3	52.1	2.8	44.5	0.0	2.1
United Kingdom								
England and Wales	1982	9th	3.0	51.1	2.6	43.7	0.2	2.5
Scotland	1984	9th	3.1	51.7	2.7	44.4	0.2	3.4
Southern Europe								
Italy	1980	9th	2.1	32.0	1.2	18.6	0.4	5.8
Portugal	1984	9th	1.8	25.6	0.8	11.9	0.6	9.2
Yugoslavia	1982	9th	1.7	28.8	0.8	13.9	0.3	4.4
Western Europe								
Austria	1984	9th	2.7	36.8	1.7	23.4	0.4	5.7
Belgium	1982	9th	2.3	34.5	1.4	20.6	0.2	3.1
France	1983	9th	2.0	24.5	0.9	11.3	0.4	4.6
Germany, Federal Republic of	1984	9th	2.8	41.8	1.9	27.9	0.3	4.9
Netherlands	1984	9th	2.9	42.1	2.1	29.9	0.2	3.1
Switzerland	1984	8th	2.6	36.9	1.7	24.2	0.2	2.9
Oceania								
Australia-New Zealand								
Australia	1983	9th	2.9	42.6	2.4	35.1	0.1	2.2
New Zealand	1983	9th	3.0	48.1	2.5	39.8	0.2	2.6
Means, 26 countries			2.7	39.4	1.8	27.0	0.3	4.3

Source: Same as table 7.

[a] The codes of the International Classification of Diseases for the causes of death shown are as follows:

	Eighth Revision (Intermediate List of 150 Causes)	Ninth Revision (Basic Tabulation List)
Diseases of circulatory system	A80-A88	25-30
Ischaemic heart disease	A83	27
Cerebro-vascular disease	A85	29

[b] The contents of the ischaemic heart disease category are not entirely comparable between the Eighth and Ninth Revisions of the ICD. See K. Uemura and Z. Pisa, "Recent trends in cardiovascular disease mortality in 27 industrialized countries", *World Health Statistics Quarterly*, vol. 38, No. 2 (1985), pp. 147-148.

females; and cancer of the male and female genital organs (see notes to table 10 for the sites included here). Of all cancer sites, lung cancer made the largest average contribution to the sex differential in life expectancy (0.8 year and 12 per cent). The largest contributions from lung cancer were in the Netherlands (1.5 years) and Belgium (1.3 years), and the smallest were in Portugal (0.3 year) and Sweden (0.4 year).

The negative contributions from the exclusively female sites of breast and female genital organs had the effect of narrowing the sex differential in life expectancy by an average of 0.7 year. The average contribution from female breast cancer was −0.4 year and ranged from −0.2 year in Japan, Romania and Yugoslavia to −0.6 year in Denmark, England and Wales (United Kingdom), the Netherlands, New Zealand and Switzerland. Mortality from cancer of the female genital organs narrowed the sex differential by 0.3 year, on average, with a range from −0.1 year in Finland, Sweden and the United States, to −0.4 year in Austria and the German Demo-

TABLE 10. CONTRIBUTION OF ALL NEOPLASMS AND OF MALIGNANT NEOPLASMS OF SELECTED SITES TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	Revision of the ICD	Contribution of neoplasms									
					Malignant neoplasms of selected sites[a]							
			All neoplasms[a]		Trachea, bronchus, lung		Female breast		Female genital organs[b]		Male genital organs[c]	
			Years	Per cent	Years	Per cent	Years	Per cent	Years	Per cent	Years	Per cent
Northern America												
Canada	1983	9th	1.3	18.0	0.9	12.6	−0.5	−7.6	−0.3	−3.9	0.4	4.9
United States	1982	9th	1.2	16.1	0.8	10.8	−0.5	−6.7	−0.1	−1.8	0.3	4.4
East Asia												
Japan	1984	9th	1.7	28.7	0.5	8.0	−0.2	−2.6	−0.2	−3.7	0.1	1.4
Europe												
Eastern Europe												
Bulgaria	1982	9th	0.7	13.2	0.6	10.3	−0.3	−4.8	−0.2	−4.4	0.1	2.0
Czechoslovakia	1982	9th	1.4	19.4	1.0	13.1	−0.3	−4.4	−0.3	−4.7	0.2	2.3
German Democratic Republic	1984	9th	1.0	16.7	0.8	13.7	−0.3	−5.5	−0.4	−6.2	0.2	3.2
Hungary	1984	9th	1.4	16.8	0.8	10.1	−0.3	−3.9	−0.3	−4.0	0.2	2.4
Poland	1984	9th	1.4	16.6	0.9	11.3	−0.3	−3.4	−0.3	−4.3	0.1	1.7
Romania	1983	9th	0.7	12.3	0.5	8.2	−0.2	−4.2	−0.3	−6.1	0.1	1.8
Northern Europe												
Denmark	1984	8th	0.8	13.5	0.6	10.7	−0.6	−9.6	−0.2	−3.4	0.3	5.7
Finland	1983	8th	1.3	15.6	1.0	12.3	−0.3	−4.2	−0.1	−1.1	0.3	3.4
Norway	1984	8th	0.9	13.6	0.5	6.8	−0.4	−6.0	−0.2	−2.2	0.4	6.3
Sweden	1984	8th	0.7	11.6	0.4	6.0	−0.4	−6.9	−0.1	−2.1	0.4	6.4
United Kingdom												
England and Wales	1982	9th	1.0	16.2	0.9	14.6	−0.6	−9.8	−0.3	−5.5	0.2	3.7
Scotland	1984	9th	0.9	15.1	0.9	14.3	−0.5	−8.9	−0.3	−5.4	0.2	3.7
Southern Europe												
Italy	1980	9th	1.7	24.8	1.0	14.2	−0.4	−6.0	−0.3	−3.9	0.2	3.0
Portugal	1984	9th	0.9	13.2	0.3	4.9	−0.3	−4.8	−0.2	−3.0	0.2	2.8
Yugoslavia	1982	9th	1.0	16.9	0.6	9.9	−0.2	−4.2	−0.3	−4.5	0.2	2.8
Western Europe												
Austria	1984	9th	1.2	16.2	0.8	10.6	−0.4	−6.1	−0.4	−5.2	0.3	3.6
Belgium	1982	9th	1.7	24.9	1.3	19.1	−0.5	−8.0	−0.3	−4.0	0.3	4.4
France	1983	9th	2.3	28.7	0.8	10.2	−0.4	−5.3	−0.3	−3.4	0.3	4.2
Germany, Federal Rep. of	1984	9th	1.3	19.1	0.8	12.0	−0.5	−6.7	−0.3	−4.7	0.3	4.5
Netherlands	1984	9th	1.9	28.0	1.5	21.4	−0.6	−8.4	−0.3	−4.3	0.4	5.3
Switzerland	1984	8th	1.7	24.5	1.0	13.8	−0.6	−8.8	−0.2	−2.5	0.5	7.3
Oceania												
Australia-New Zealand												
Australia	1983	9th	1.3	18.7	0.8	11.2	−0.5	−6.7	−0.3	−3.8	0.3	4.9
New Zealand	1983	9th	0.8	12.8	0.7	11.4	−0.6	−9.2	−0.3	−5.3	0.3	5.1
Means, 26 countries			1.2	18.1	0.8	11.6	−0.4	−6.3	−0.3	−4.0	0.3	3.9

Source: Same as table 7.

[a] The figures for "All neoplasms" include malignant, benign and unspecified neoplasms. The figures for neoplasms of selected sites are for malignant neoplasms only. The codes of the International Classification of Diseases for the causes of death shown are as follows:

	Eighth Revision (Intermediate List of 150 Causes)	Ninth Revision (Basic Tabulation List)
All neoplasms	A45-A61	08-17
Malignant neoplasms:		
Trachea, bronchus, lung	A51	101
Female breast	A54	113
Female genital organs	A55-A56	120-123
Male genital organs	A57	124-125

[b] The figures for "Female genital organs" for the Ninth Revision of the ICD include malignant neoplasms of the cervix uteri; placenta; other uterus; and ovary and other uterine adnexa. The figures for the Eighth Revision of the ICD include all the aforementioned sites except "ovary and other uterine adnexa".

[c] The figures for "Male genital organs" for the Ninth Revision of the ICD include malignant neoplasms of the prostate and testis, but for the Eighth Revision of the ICD, the figures pertain only to the prostate.

cratic Republic. The positive contribution from cancer of the male genital organs also averaged 0.3 year, so the net effect on the sex differential from male and female genital cancer

The contribution from stomach cancer, not shown separately in table 10, was only 0.1 or 0.2 year in all countries but Japan (where it was 0.5 year) and Poland (0.3 year).

was 0.

As mentioned above, accidents, suicide and violence together contributed an average of 1.3 years and 19 per cent to the sex differential in life expectancy. Although there were substantial country-to-country variations, the

TABLE 11. CONTRIBUTION OF EXTERNAL CAUSES (ACCIDENTS, SUICIDE, VIOLENCE) TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, LATEST AVAILABLE YEAR

Region and country	Year	Revision of the ICD	Contribution of external causes[a]									
			All external causes		Motor vehicle accidents		Other accidents		Suicide		Homicide	
			Years	Per cent	Years	Per cent	Years	Per cent	Years	Per cent	Years	Per cent
Northern America												
Canada	1983	9th	1.4	19.4	0.4	6.0	0.4	5.9	0.4	6.0	0.0	0.4
United States	1982	9th	1.7	22.1	0.5	6.7	0.4	5.5	0.3	4.5	0.3	3.8
East Asia												
Japan	1984	9th	1.1	18.8	0.3	5.6	0.4	5.9	0.4	6.6	0.0	0.0
Europe												
Eastern Europe												
Bulgaria	1982	9th	1.3	24.3	0.3	5.8	0.7	12.7	0.2	4.1	0.1	1.0
Czechoslovakia	1982	9th	1.4	18.6	0.3	3.8	0.6	8.2	0.4	5.7	0.0	0.2
Hungary	1984	9th	1.8	22.5	0.4	4.6	0.6	7.3	0.8	9.8	0.0	0.4
Poland	1984	9th	1.9	23.3	0.4	5.5	0.8	9.8	0.4	5.2	0.0	0.3
Northern Europe												
Denmark	1984	8th	0.9	15.3	0.3	4.3	0.2	3.8	0.4	6.4	0.0	0.1
Finland	1983	8th	1.8	21.7	0.2	2.9	0.8	9.0	0.7	8.4	0.0	0.4
Norway	1984	8th	1.2	18.0	0.3	4.7	0.5	7.2	0.4	5.5	0.0	0.5
Sweden	1984	8th	1.1	18.3	0.3	4.5	0.3	5.1	0.4	6.0	0.0	0.2
United Kingdom												
England and Wales	1982	9th	0.6	10.6	0.3	4.6	0.2	2.9	0.1	2.4	−0.0	−0.1
Scotland	1984	9th	0.8	13.9	0.3	4.8	0.3	4.5	0.2	3.4	0.0	0.4
Southern Europe												
Italy	1980	9th	1.0	15.2	0.5	8.0	0.3	4.1	0.1	1.7	0.1	0.8
Portugal	1984	9th	1.8	25.6	0.8	12.1	0.5	7.6	0.2	2.7	0.0	0.7
Yugoslavia	1982	9th	1.4	23.5	0.6	9.6	0.5	8.6	0.3	4.6	0.0	0.7
Western Europe												
Austria	1984	9th	1.8	25.3	0.7	10.1	0.5	6.6	0.6	8.1	0.0	0.1
Belgium	1982	9th	1.2	18.4	0.5	8.0	0.3	4.5	0.3	5.1	0.0	0.2
France	1983	9th	1.6	19.4	0.5	6.5	0.5	6.1	0.4	5.5	0.0	0.2
Germany, Federal Rep. of	1984	9th	1.0	14.8	0.4	5.6	0.2	3.4	0.4	5.3	−0.0	−0.0
Netherlands	1984	9th	0.6	9.1	0.3	4.1	0.1	2.2	0.1	2.1	0.0	0.4
Switzerland	1984	8th	1.4	20.7	0.5	7.0	0.3	4.8	0.5	7.9	0.0	0.2
Oceania												
Australia-New Zealand												
Australia	1983	9th	1.2	18.0	0.5	7.3	0.4	5.6	0.3	4.3	0.0	0.3
New Zealand	1983	9th	1.2	18.8	0.5	7.4	0.4	6.8	0.2	3.7	0.0	0.5
Means, 24 countries			1.3	19.0	0.4	6.2	0.4	6.2	0.4	5.2	0.0	0.5

Source: Same as table 7.

[a] The codes of the International Classification of Diseases for the causes of death shown are as follows:

	Eighth Revision (Intermediate List of 150 Causes)	Ninth Revision (Basic Tabulation List)
External causes	AE138-AE150	E47-E56
Motor vehicle accidents	AE138	Part of E47 (E471)
Other accidents	AE139-AE146	Remainder of E47 (E470, E472-E474)
Suicide	AE147	E54
Homicide	AE148	E55
Other violence	AE149-AE150	E56

average contributions for motor vehicle accidents, other accidents and suicide were similar: 0.42 year, 0.41 year and 0.35 year, respectively (table 11). The range of contributions from motor vehicle accidents was from 0.2 year in Finland to 0.8 year in Portugal; for other accidents, from 0.1 year in the Netherlands to 0.8 year in Poland; for suicide, from 0.1 year in England and Wales, Italy and the Netherlands, to 0.8 year in Hungary. The contribution to the sex differential from homicide was negligible, except in the United States, where it contributed 0.3 year (3.8 per cent).

Although data for chronic liver disease and cirrhosis have not been presented separately, these conditions, often associated with alcohol consumption, contributed an average of 0.2 year to the sex differential in life expectancy, but as much as 0.5 or 0.6 year in Austria, Hungary, Italy and Portugal.

III. TRENDS SINCE THE NINETEENTH CENTURY

A. *Trends in the sex differential in life expectancy*

Data for the nineteenth and early twentieth centuries (table 12) show sex differentials in life expectancy at birth as small as half a year in several countries (Bulgaria, Ireland, Italy and Japan). By the early 1980s, the sex differential in life expectancy averaged about 6.7

years in the developed countries. The period when sex differentials began to widen varies among countries. Fluctuations in the sex differential, without a clear trend, occur in several countries in the nineteenth and early twentieth centuries (table 12). In Norway, for example, the differential fluctuates between 2.6 and 3.0 years in the period from 1856-1865 to 1920, while in Australia, the differential varies from 3.6 to 4.1 years in the period 1881-1890 to 1932-1934. Spain also shows little change in the sex differential in life expectancy from 1900 to 1920, when it varies from 1.6 to 1.8 years.

In several countries, the early data show a narrowing of the sex differential. Such declines are seen in Denmark (from 3.3 years in 1901-1905 to 1.6 years in 1921-1925), Sweden (from 3.9 years in 1851-1860 to 2.4 years in 1930) and the Netherlands (from 2.5 years in 1900-1909 to 1.5 years in 1931-1940). The decline shown for the United States, from a differential of 3.4 years in 1909-1911 to 2.2 years in 1919-1921, is not an artifact of the increasing death-registration area (described in a note to table 12), but reflects a real narrowing of the sex differential in life expectancy. Had the population coverage been the same at both dates, however, the magnitude of the trend might have differed somewhat.

Countries in which a decided upward trend is already evident in the early decades of the table 12 data include Japan (from 0.4 to 2.7 years between 1899-1903 and 1935/36), Ireland (from 0.3 to 1.5 years between 1900-1902 and 1935-1937), Finland (from 2.8 years in 1881-1890 to 4.8 years in 1931-1933) and France (from 2.1 to 4.7 years between 1875-1877 and 1928-1933).

By the 1930s, sex differentials of four years or more were seen in the USSR (6 years), Finland (4.8 years), France (4.7 years), Portugal (4.4 years), Switzerland (4.3 years), and Austria and England and Wales (4.1 years each).

From the 1930s to the 1970s, increases in the sex differential in life expectancy were a universal feature of mortality trends in developed countries. (The decade of the 1940s has been excluded from the analysis because of possible distortions of trends owing to heavy war casualties and large population transfers.) Of the 26 populations with data for the 1930s in table 12, the largest increases in the sex differential in life expectancy between the 1930s and 1970-1974 were in Canada (5.2 years), Northern Ireland (5.0 years), the Netherlands (4.5 years), the United States (4.1 years) and Italy (4.1 years). Of the remaining countries, in 12 the sex differential widened by between two and three years from the 1930s to 1970-1974. Countries varied as to whether the increase was more rapid between the 1930s and the 1950s or between the 1950s and the 1970s. The averages for the 26 populations show an increase in the sex differential in life expectancy of 1.4 years between the 1930s and 1950-1954, and 2.0 years between 1950-1954 and 1970-1974.

From 1970-1974 on, countries fall into three groups based on their trends in the sex differential in life expectancy: those in which the differential appears to be narrowing, after reaching its highest level in the 1970s or early 1980s; countries where the differential is neither increasing nor decreasing; and countries in which the most recent data indicate a still widening differential. A substantial number of countries fall into the first two groups, suggesting a departure from the trends of the preceding four decades, when differentials were widening in all developed countries.

The inclusion of annual data for the 1980s in table 12 may help to signal incipient trends that are not yet apparent from the five-year averages. However, annual data are subject to greater random fluctuations, as well as to fluctuations associated with influenza epidemics. Any conclusions drawn from the annual data must therefore be tentative.

The United Kingdom was among the first to show a narrowing of the sex differential: in England and Wales, the differential narrowed from 6.3 years in 1970-1974 to 5.9 years in 1983, while in Scotland the decline was from 6.4 years in 1970-1974 and 1975-1979 to 5.8 years in 1985. The figures for Northern Ireland are more erratic, but a slight narrowing of the differential may be under way.

Other countries where the sex differential in life expectancy has been narrowing include Canada, where it reached 7.6 years in 1975-1979, and subsequently declined to 7.1 years in 1984; the United States, where the sex differential declined from 7.9 years in 1975-1979 to 7.3 years in 1983; and Finland, where it has declined from 8.9 years in 1975-1979 to 8.5 years in 1985. A slight narrowing is also observed in Portugal (from 7.2 years in 1975-1979 to 7.0 years in 1980-1984). The official life tables for the USSR show a decline from 10 years in 1971-1972 to 9 years in 1985/86.

The five-year averages for Australia for 1975-1979 and 1980-1984 indicate a sex differential of 7.0 years in both periods, but the annual data for the 1980s show a fairly steady decline to 6.7 years by 1984. A decline from 6.5 years in 1975-1979 to 6.2 years in 1980-1984 is seen for New Zealand, although the annual data for the 1980s are erratic.

The sex differential appears to have stabilized (at least temporarily) in nine countries. In several of those countries, however, the trends are fairly recent and are indicated by the annual data for the 1980s. The conclusions are therefore tentative. In the Federal Republic of Germany, the highest quinquennial sex differential in life expectancy—6.7 years—was reached in 1975-1979, and the differential has remained at that level in most years through 1985. The sex differential in the German Democratic Republic was virtually unchanged from 1975-1979 (5.8 years) to 1980-1984 (5.9 years), and the annual data for the 1980s no longer show an increasing trend, but fluctuate between 5.8 and 6.0 years. A similar pattern is observed in Denmark and France, where the sex differential increased by only 0.1 year between 1975-1979 and 1980-1984, and the annual data for the 1980s show a stable differential that varies between 6.8 and 7.0 years in Denmark and between 8.2 and 8.4 years in France. In Norway, the differential increased from 6.5 to 6.9 years between 1975-1979 and 1980-1984, but a levelling off is evident in the annual data for the 1980s, which

TABLE 12. TRENDS IN LIFE EXPECTANCY AT BIRTH (BOTH SEXES) AND THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, DEVELOPED COUNTRIES, NINETEENTH CENTURY TO 1980s

(*Years*)

Region and country	*Item*[b]	*Period*[a]														
		Nineteenth century	*1900s*	*1910s*	*1920s*	*1930s*	*1950 to 1954*	*1960 to 1964*	*1970 to 1974*	*1975 to 1979*	*1980 to 1984*[c]	*1980*	*1981*	*1982*	*1983*	*1984*	*1985*
Americas																	
Northern America																	
Canada	e_0				58.8	61.1	69.1	71.4	73.1	74.3	75.8	75.2	75.6	75.5	76.2	76.6	
	Diff. e_0				2.0	2.1	4.7	6.0	7.3	7.6	7.2	7.4	7.4	7.0	7.2	7.1	
United States[d]	e_0		49.7	52.0	57.5	61.0	68.9	70.2	71.4	73.4	74.4	73.9	74.3	74.6	74.7		
	Diff. e_0		2.8	3.4	2.2	3.6	5.9	6.7	7.7	7.9	7.5	7.7	7.6	7.4	7.3		
East Asia																	
Japan	e_0		38.0	40.5	45.7	48.3	62.2	69.2	73.3	75.4	77.1	76.3	76.7	77.3	77.4	77.8	78.0
	Diff. e_0		0.4	0.6	1.7	2.7	3.5	5.0	5.4	5.3	5.7	5.5	5.5	5.7	5.8	5.9	6.0
Europe																	
Eastern Europe																	
Bulgaria	e_0		40.0		46.3	49.8	66.7	70.2	71.2	71.1	71.4	71.2	71.7	71.3	71.4	71.5	
	Diff. e_0		0.6		0.7	1.1	4.0	3.7	4.7	5.1	5.7	5.4	5.5	5.6	6.0	6.0	
Czechoslovakia	e_0		40.3	43.7		53.6	69.1	70.5	70.0	70.6	70.8	70.6	70.8	70.9	70.6	70.9	
	Diff. e_0		2.8	3.0		3.2	5.1	5.7	7.0	7.2	7.4	7.3	7.5	7.4	7.4	7.5	
German Democratic Republic	e_0	-	-	-	-	-	66.1	69.8	71.2	71.7	72.1	71.7	71.9		72.4	72.5	
	Diff. e_0	-	-	-	-	-	4.4	4.9	5.5	5.8	5.9	6.0	5.9		6.0	5.8	
Hungary[e]	e_0		37.4		42.1	49.8	63.6	68.7	69.5	69.6	69.2	69.1	69.3	69.4	69.1	69.2	69.2
	Diff. e_0		1.6		2.1	3.0	4.3	4.6	5.9	6.4	7.7	7.3	7.5	7.6	8.0	8.2	8.1
Poland	e_0					49.8	60.7	68.1	70.6	70.9	71.0	70.4	71.3	71.3	71.2	70.9	
	Diff. e_0					3.3	5.6	6.1	7.1	8.0	8.2	8.5	8.3	8.0	8.2	8.2	
Romania	e_0							66.9	68.9	69.7	69.6	69.2	69.6	69.7	69.7	69.9	
	Diff. e_0							3.9	4.5	4.8	5.5	5.3	5.6	5.4	5.6	5.6	
Northern Europe																	
Denmark	e_0		54.7	57.8	61.2	63.0	71.0	72.5	73.7	74.4	74.6	74.2	74.5	74.8	74.6	74.8	
	Diff. e_0		3.3	3.0	1.6	1.8	2.6	4.0	5.5	5.9	6.1	6.1	6.2	6.1	6.2	6.0	
Finland	e_0	42.8	45.6		52.9	56.3	66.1	69.0	70.8	72.5	74.2	73.6	73.9	74.5	74.4	74.8	
	Diff. e_0	2.8	2.9		4.4	4.8	6.5	7.0	8.5	8.9	8.6	8.8	8.6	8.6	8.3	8.5	
Ireland	e_0		49.5	53.9	57.7	59.1	66.7	70.1	71.1	71.9	72.9	72.5	72.9		73.2		
	Diff. e_0		0.3	0.5	0.6	1.5	2.7	3.8	4.9	5.3	5.6	5.3	5.7		5.7		
Norway	e_0	48.8	55.4	57.8	59.3	64.2	72.7	73.4	74.5	75.4	76.1	75.9	76.0	76.0	76.3	76.4	
	Diff. e_0	2.6	3.0	2.9	2.8	3.2	3.5	4.8	6.3	6.5	6.9	7.0	6.8	7.0	7.0	6.8	
Sweden	e_0	42.5	55.8	58.1	58.9	63.2	71.7	73.5	74.9	75.4	76.5	75.9	76.2	76.5	76.7	77.0	
	Diff. e_0	3.9	2.5	2.9	2.7	2.4	2.8	4.0	5.5	6.2	6.2	6.2	6.2	6.1	6.1	6.2	
United Kingdom																	
England and Wales	e_0	43.0	50.5	53.5	57.6	60.8	69.5	71.2	72.2	73	74.2	73.7	74.2	74.3	74.5		
	Diff. e_0	3.3	3.9	3.8	3.9	4.1	5.2	6.0	6.3	6.1	6.0	6.0	6.0	5.9	5.9		
Northern Ireland	e_0				55.8	58.5	67.7	70.2	70.4	71.1	72.4	71.7	72.7	72.9			73.4
	Diff. e_0				0.7	1.4	3.6	4.9	6.4	6.4	6.3	6.5	6.6	5.7			6.2
Scotland	e_0			51.6	54.8	57.7	67.2	69.2	70.5	71.2	72.4	71.9	72.2	72.2	72.7	72.9	73.0
	Diff. e_0			3.0	3.3	3.5	4.5	5.9	6.4	6.4	6.2	6.4	6.3	6.1	6.1	6.0	5.8
Southern Europe																	
Greece	e_0						70.2	71.8	74.1	74.9	75.7	75.3	75.3	75.9		76.2	
	Diff. e_0						2.9	3.6	4.3	4.6	4.7	4.5	4.6	4.7		4.8	
Italy	e_0	35.4	43.1	47.0	49.6	54.5	66.4	69.7	72.1	73.6	74.6	74.3	74.9				
	Diff. e_0	0.5	0.3	0.8	1.5	2.1	3.7	5.3	6.2	6.5	6.7	6.7	6.7				
Portugal	e_0				38.0	47.1	60.0	64.4	67.8	69.7	71.9	71.1	71.1	72.6		72.9	73.1
	Diff. e_0				4.2	4.4	5.1	5.7	6.4	7.2	7.0	7.1	7.0	7.0		7.0	7.1

Spain	e_0		34.8	41.8	41.2	50.4	60.9	71.2	72.5	74.1		75.5					
	Diff. e_0		1.8	1.6	1.8	3.2	4.8	5.3	5.6	6.0		6.2					
Yugoslavia	e_0						57.7	64.7	68.4	70.2	70.6	70.6	70.5	70.8			
	Diff. e_0						3.2	3.4	4.9	5.2	5.7	5.5	5.6	5.9			
Western Europe																	
Austria	e_0		38.8			56.6	66.2	69.5	70.5	71.8	73.0	72.6	72.8	73.0	73.1	73.7	73.9
	Diff. e_0		2.1			4.1	5.3	6.4	7.2	7.1	7.2	7.1	7.2	7.3	7.1	7.2	7.0
Belgium	e_0	45.2				58.0	67.7	70.3	71.4	72.6	73.7	73.2	73.6	73.8		74.3	
	Diff. e_0	3.0				3.7	5.1	6.0	6.5	6.6	6.9	6.8	6.9	6.8		7.0	
France	e_0	43.0	47.0	50.5	54.1	58.8	67.5	71.2	73.1	74.2	75.3	74.9	75.0	75.4	75.3	75.9	
	Diff. e_0	2.1	3.4	3.9	3.9	4.7	5.9	6.8	7.8	8.2	8.3	8.3	8.2	8.3	8.2	8.4	
Germany, Federal Republic of.	e_0	-	-	-	-	-	67.3	70.9	70.9	72.3	73.9	73.4	73.6	73.9	74.1	74.7	75.0
	Diff. e_0	-	-	-	-	-	4.2	3.7	6.5	6.7	6.7	6.8	6.7	6.7	6.7	6.7	6.7
Netherlands	e_0	39.6	52.2	56.1	62.8	66.5	72.0	73.6	74.1	75.2	76.2	76.0	76.1	76.2	76.4	76.5	
	Diff. e_0	2.3	2.5	2.0	1.4	1.5	2.5	4.6	6.0	6.6	6.9	6.9	6.8	6.9	6.8	6.9	
Switzerland	e_0	41.9	50.7		59.8	61.3	69.4	71.6	73.8	75.3	76.3	75.7	75.9	76.3	76.3	77.3	77.0
	Diff. e_0	2.6	2.9		3.3	4.3	4.7	5.8	6.2	6.7	6.8	6.7	6.8	6.7	7.1	6.9	6.9
Oceania																	
Australia-New Zealand																	
Australia	e_0	49.1	57.0	59.6	61.3	65.4	69.4	71.0	71.4	73.5	75.2	74.6	75.1	74.8	75.6	76.0	
	Diff. e_0	3.7	3.6	3.8	4.1	3.6	5.5	6.3	6.9	7.0	7.0	7.1	7.2	7.0	6.8	6.7	
New Zealand[f]	e_0	55.9	59.3	62.2	65.4	67.0	69.7	71.2	71.7	72.5	73.7	72.8	73.7	73.8	73.9	74.5	
	Diff. e_0	2.9	2.4	2.4	2.5	3.0	4.3	5.5	6.2	6.5	6.2	5.7	6.5	6.2	6.2	6.6	
USSR[g]	e_0	32.0			44.0	47.0	67.0		70.0						68	68	69.0
	Diff. e_0	2.0			5.0	6.0	6.0		10.0						10	10	9.0

Sources: For dates before 1950, the values shown are mainly from the country life tables that served as a basis for the Coale-Demeny regional model life tables. For pre-1950 values that have been taken from other sources, those sources are indicated opposite the country name in the list below.

For the 1950s to the 1980s, most values are from life tables calculated by the United Nations from the World Health Organization data bank of mortality and population statistics. Some of the values for the most recent years are from *World Health Statistics Annual 1985* (Geneva, 1985), table 5; and *World Health Statistics Annual 1986* (Geneva, 1986), table 10. For post-1950 data that have been taken from other sources, those sources are indicated by country in the list below.

Australia, 1911: S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972).

Bulgaria, 1906-1912 and 1933-1936: N. Naoumov, I. Stefanov and Z. Sougarev, *La population de la Bulgarie*, CICRED Series, Monograph for World Population Year 1974 (Sofia, 1974), pp. 18, 98; 1925-1928: official life table.

Czechoslovakia, 1909-1912: V. Srb, *La population de la Tchécoslovaquie*, CICRED Series, Monograph for World Population Year 1974 (Committee for International Co-operation in National Research in Demography, 1975), p. 16.

Finland, 1901-1905: Finland, Central Statistical Office, *Cohort Mortality in Finland from 1851* (Helsinki, 1980), p. 59; 1921-1930: Finland, Population Research Institute, *Yearbook of Population Research in Finland 1982* (Helsinki, 1982), p. 94.

Hungary, 1901-1902 and 1920-1921: E. Szabady, *The Population of Hungary*, CICRED Series, Monograph for World Population Year 1974 (1975), p. 46.

Ireland, 1900-1902 and 1910-1912: *Demographic Yearbook 1957* (United Nations publication, Sales No. 57.XIII.1), table 24.

Italy, 1921 and 1931: Preston, Keyfitz and Schoen, *op. cit.*

Japan, 1899-1903 and 1909-1913: H. Mizushima, *Seimeihyo no kenkyu (Study of the Life Table)* (Osaka, Semeihoken-bunka-kenkyujo, 1963), p. 127. Mizushima's life tables adjust for underregistration of deaths, and therefore show lower life expectancies than the official life tables for the same periods; for 1926-1930 and 1935-1936: *Demographic Yearbook 1957* . . .

Netherlands, 1910-1920: Netherlands, Central Bureau voor de Statistiek, *Sterftetafels voor Nederland; Afgeleid uit Waarnemingen over de Periode 1971-1975* ('s-Gravenhage, Staatsuitgeverij, 1977).

Norway, 1901-1905: Calculated from death rates in Norway, Statistisk Sentralbyra, *Historisk Statistikk 1978* (Oslo, 1978), pp. 54-55, and Norway, Statistisk Sentralbyra, *Dodeligheten og dens Arsaker i Norge, 1856-1955* (Oslo, 1961); for 1910, 1920 and 1930: Preston, Keyfitz and Schoen, *op. cit.*

Sweden, 1901-1910: *Demographic Yearbook 1957*. . . ; for 1911, 1920 and 1930: Preston, Keyfitz, and Schoen, *op. cit.*

Switzerland, 1876-1880, 1901-1910 and 1921-1930: J. Cipriani and others, *La population de la Suisse*, CICRED Series, Monograph for World Population Year 1974 (Berne, 1974), p. 46.

USSR: All data are from USSR, Central Statistical Board, *Narodnoye khoziastvo SSSR v 1986 godu* (Moscow, 1986), p. 547.

United Kingdom (Northern Ireland), 1936-1938: *Demographic Yearbook, 1957*. .

NOTE: A blank in a cell does not necessarily mean that data for the period indicated are not available, as the data in this table are not meant to be exhaustive. See the explanation under "Sources" of which data were included.

[a] The exact time periods for dates before 1950 are given in the annex, and for dates after 1950 if different from dates indicated in column headings.

[b] "e_0" is the mean life expectancy at birth for both sexes; "Diff. e_0" is the difference between the female and male life expectancy at birth.

[c] Values may differ slightly from those shown in earlier tables because of the inclusion in this table of more recent data for some countries.

[d] Data are for a changing death-registration area, which covered 26 per cent of the total population of the United States in 1900, 51 per cent in 1910, 81 per cent in 1920, and 95 per cent in 1930; 100 per cent coverage was achieved in 1933. See United States, Department of Commerce, Bureau of the Census, *Historical Statistics of the United States, Colonial Times to 1970, Part 1* (Washington, D.C., Government Printing Office, 1975), p. 44. The early life tables are not representative of the entire country. As a condition for the admission of states to the death-registration area, death registration had to be at least 90 per cent complete in the state, and completeness of registration is generally associated with favourable mortality. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972), p. 29. Apparent mortality changes in successive life tables before 1933 are therefore affected by the increasing geographic coverage of the population.

[e] For 1900-1901, excluding Croatia-Slavonia.

[f] Prior to 1950, data are for white population only.

[g] For 1896-1897, data are for 50 governorates of European Russia; for 1926-1927, for the European part of the USSR.

vary between 6.8 and 7.0 years. The patterns in Belgium and the Netherlands are almost the same as in Norway: an increase in the differential from 6.6 years (1975-1979) to 6.9 years (1980-1984), with slight fluctuations in the annual data for the 1980s. Finally, in Poland, although the sex differential in life expectancy increased from an average of 8.0 years (1975-1979) to 8.2 years (1980-1984), the annual values for the 1980s show a narrowing from 8.5 years (1980) to 8.2 years (1984), suggesting a stabilization of the differential, or perhaps even the start of a declining trend.

Recent data show that sex differentials are still increasing in about half the countries. Pronounced upward trends are evident in Hungary (an increase from 7.3 years in 1980 to 8.1 years in 1985), Japan (from 5.5 to 6.0 years over the same period) and Bulgaria (from 5.4 to 6.0 years between 1980 and 1984). Increases of 0.4 year between 1980 and 1983 are seen in Ireland and Yugoslavia, while in Italy and Spain an increase of 0.2 year occurred between 1975-1979 and 1980 or 1981. The sex differential in Romania increased from 4.8 to 5.5 years between 1975-1979 and 1980-1984. The annual data for the 1980s show the differential still increasing, but at a slower pace. Of the aforementioned countries, Bulgaria, Ireland, Japan, Romania and Yugoslavia ranked among the lowest in terms of size of sex differential in life expectancy in the early 1980s (see table 2), and now appear to be "catching up".

The preceding discussion of trends was based on the absolute size of the difference between the male and female life expectancies at birth. Had a relative measure been employed, such as the sex differential in life expectancy taken as a percentage of the mean life expectancy at birth for both sexes, different results would have been obtained with regard to the onset and the pace of widening of the sex differential.

B. *Some features of the transition from high to low mortality*

The widening of the sex differential in life expectancy is only one aspect of the profound mortality changes of the past century. Some of those changes are illustrated by the data for England and Wales and Italy in tables 13, 14 and 15. While the early cause-of-death data for England and Wales are believed to be among the most reliable of any country for the late nineteenth and early twentieth centuries, the Italian data are poorer in quality, as evidenced by the large percentage of deaths in "all other causes". Neither country can be considered as having had a "typical" pattern of mortality in the early data. Preston comments about England and Wales: "Unfortunately, the country with the most satisfactory early data appears to offer an atypical account of mortality decline, a record that may be largely responsible for prevailing representations of mortality reduction" (Preston, 1976, p. 20).

In England and Wales, life expectancy at birth (both sexes) increased from 47 years in 1901 to 74 years in 1982, while Italy showed an even larger gain, from 43 years to 74 years between 1901 and 1980 (table 13, part A). These gains reflect large declines in age-specific mortality. However, all age groups did not benefit equally from the mortality declines. With a few exceptions, there was a general pattern for mortality improvement to vary inversely with age. Percentage declines were greatest in age groups under 10 years (table 14). At

TABLE 13. SOME FEATURES OF THE TRANSITION FROM HIGH TO LOW MORTALITY, ILLUSTRATED BY DATA FOR ENGLAND AND WALES AND ITALY: CHANGES IN LIFE EXPECTANCY AND IN THE AGE STRUCTURE OF MORTALITY

	1901			*1980s*[a]		
	Both sexes	*Males*	*Females*	*Both sexes*	*Males*	*Females*
A. *Life expectancy at birth (years)*						
Country						
England and Wales	47.38	45.32	49.43	74.28	71.31	77.25
Italy	43.34	43.03	43.65	74.31	70.97	77.66
B. *Percentage distribution of deaths by age group, England and Wales*						
Age group						
All ages	100.0	100.0	100.0	100.0	100.0	100.0
0-4	36.6	38.4	34.7	1.4	1.6	1.1
5-14	4.0	3.8	4.2	0.3	0.3	0.2
15-24	4.4	4.4	4.4	0.8	1.2	0.4
25-34	5.5	5.4	5.6	0.8	1.1	0.6
35-44	7.0	7.2	6.8	1.6	1.9	1.3
45-54	8.4	8.9	7.9	4.5	5.6	3.4
55-64	10.5	10.7	10.4	12.8	16.0	9.5
65-74	12.1	11.4	12.8	26.3	31.4	21.3
75+	11.6	9.9	13.4	51.6	41.0	62.1

Sources: For 1901, calculated from S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972); for the 1980s, calculated from the World Health Organization data bank.

NOTE: Sums of detail do not in all cases equal totals, because of rounding.

[a] Data refer to 1982 for England and Wales and to 1980 for Italy.

TABLE 14. SOME FEATURES OF THE TRANSITION FROM HIGH TO LOW MORTALITY, ILLUSTRATED BY DATA FOR ENGLAND AND WALES AND ITALY: DECLINES IN AGE-SPECIFIC MORTALITY

Age group (years)	Age-specific death rates (per 1,000 population)								Percentage decline in death rates			
	England and Wales				Italy				England and Wales		Italy	
	Males		Females		Males		Females		1901-1982		1901-1980	
	1901	1982	1901	1982	1901	1980	1901	1980	Males	Females	Males	Females
0	175.4	12.2	141.6	9.4	175.6	16.4	158.2	12.4	−93.1	−93.4	−90.7	−92.1
1-4	21.3	0.5	20.4	0.4	36.0	0.5	37.2	0.5	−97.6	−98.0	−98.5	−98.7
5-9	4.0	0.3	4.1	0.2	5.7	0.4	6.3	0.3	−93.8	−95.8	−93.7	−95.6
10-14	2.3	0.3	2.4	0.2	3.4	0.4	4.5	0.3	−87.8	−92.0	−88.2	−94.2
15-19	3.5	0.8	3.2	0.3	4.5	1.0	4.4	0.4	−77.1	−91.0	−77.9	−92.0
20-24	4.7	0.9	3.8	0.4	5.5	1.1	6.1	0.4	−81.6	−90.9	−79.7	−93.5
25-29	5.5	0.8	4.7	0.4	6.3	1.0	6.9	0.4	−85.0	−91.1	−84.2	−93.8
30-34	7.1	1.0	6.0	0.6	7.3	1.1	8.0	0.6	−86.3	−89.7	−84.6	−92.4
35-39	9.2	1.3	7.7	0.9	8.8	1.7	9.6	0.9	−86.0	−88.1	−80.5	−90.3
40-44	12.2	2.3	9.9	1.5	9.4	2.7	7.8	1.4	−81.1	−84.6	−71.5	−82.1
45-49	15.5	4.1	11.8	2.7	12.0	4.9	9.9	2.3	−73.3	−77.2	−59.7	−76.2
50-54	21.2	7.6	16.3	4.5	15.7	8.5	13.3	3.6	−64.1	−72.1	−46.2	−72.8
55-59	27.9	13.6	21.8	7.5	20.6	13.6	18.1	5.8	−51.2	−65.5	−34.1	−67.8
60-64	40.4	21.7	32.1	11.8	37.7	21.4	36.7	9.4	−46.3	−63.2	−43.2	−74.4
65-69	55.6	36.0	45.8	18.6	53.1	33.1	52.3	15.7	−35.3	−59.4	−37.7	−70.1
70-74	85.9	57.7	71.8	30.5	80.1	50.5	79.1	27.8	−32.9	−57.5	−36.9	−64.9
75-79	115.7	90.1	100.6	51.3	123.0	78.7	122.0	50.0	−22.2	−49.0	−36.0	−59.0
80-84	192.1	137.9	166.5	88.5	204.5	128.1	197.1	96.4	−28.2	−46.9	−37.4	−51.1
85+	277.0	223.2	248.7	178.2	379.2	220.6	365.9	183.5	−19.4	−28.3	−41.8	−49.8

Sources: For 1901, calculated from S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972); for the 1980s, calculated from the World Health Organization data bank.

ages 1-4, death rates fell by 98 or 99 per cent between the turn of the century and the 1980s, followed by declines of 94 to 96 per cent at ages 5-9, and 91 to 93 per cent during infancy (age 0). In these three youngest age groups, the declines in mortality were of similar magnitude for both sexes.

At older ages, mortality improvement has generally been much greater for females than for males. This can be seen in the data for England and Wales and Italy in the following table:

MEAN PERCENTAGE DECLINES IN AGE-SPECIFIC MORTALITY, ENGLAND AND WALES AND ITALY

Age group	England and Wales 1901-1982		Italy 1901-1980	
	Males	Females	Males	Females
20-24 to 40-44	−84.0	−88.9	−80.1	−90.4
45-49 to 60-64	−58.7	−69.5	−45.8	−72.1
65-69 to 70-74	−34.1	−58.5	−37.3	−67.5
75-79 to 80-84	−25.2	−48.0	−36.7	−55.1

Source: Data are simple means of the percentage declines for the more detailed age groups in table 14.

These data also show that the divergencies between the sexes with regard to mortality decline are much more pronounced for Italy than for England and Wales in the age range from 20-24 years through 70-74 years. Italian females had only a 0.6-year advantage over Italian males in 1901, but a 6.7-year advantage in 1980.

The structure of mortality by age groups has also changed radically (table 13, part B). Mortality has shifted from the very young ages to the very old. In England and Wales in 1901, about one third of all deaths occurred to children under five years old, and one third to persons over 55. The corresponding figures in 1982 were 1 per cent of total deaths to children under five, and about 90 per cent to persons over 55. The changing age distribution of deaths reflects the large declines in age-specific mortality at the young ages, as mentioned above, but it also reflects the change to an older age structure of the population in developed countries as a consequence of fertility and mortality declines.

In the course of the transition to low mortality, the structure of mortality by cause has been transformed. In table 15, causes of death have been grouped into two major groups. Group I includes causes that are generally important in high-mortality populations: the infectious and parasitic diseases; acute respiratory diseases; diarrhoeal disease; maternal mortality; and certain diseases of infancy. Group II includes the two major cause-of-death groups in low-mortality countries: neoplasms and diseases of the circulatory system. The age-standardized death rates for England and Wales in 1861, 1901 and 1982 are shown for groups I and II, as well as for their components. Changes in the cause structure of mortality include large declines in the percentage of deaths attributable to group I causes (for males, from 44 per cent of all deaths in 1861 to 14 per cent in 1982; for females, from 46 per cent to 13 per cent in the same period). The increase in the percentage of total deaths from group II causes was much greater: from about 15 to about 70 per cent for each sex between 1861 and 1982. It is important to note, however, that the validity of the comparison is affected by the large percentage of deaths classified as "All other causes" in 1861 (36 per cent) and 1901 (25 per cent), compared with 1982 (only 11 per cent). A high percentage of deaths not classified by cause is a common feature of mortality statistics in high-mortality

TABLE 15. SOME FEATURES OF THE TRANSITION FROM HIGH TO LOW MORTALITY: CHANGES IN STRUCTURE OF MORTALITY BY CAUSE, ENGLAND AND WALES, 1861-1982

Cause of death	Males						Females					
	Age-standardized death rates[a] (per 100 000 population)			Percentage distribution			Age-standardized death rates[a] (per 100 000 population)			Percentage distribution		
	1861	1901	1982	1861	1901	1982	1861	1901	1982	1861	1901	1982
All causes	2 612	2 348	968.3	100.0	100.0	100.0	2 336	1 971	575.8	100.0	100.0	100.0
Group I	1 138	992	140.0[b]	43.6	42.2	14.4[b]	1 077	816	71.5[b]	46.0	41.4	12.5[b]
Infectious and parasitic diseases	565	364	4.2	21.6	15.5	0.4	559	275	2.6	23.9	14.0	0.5
Pneumonia, influenza, bronchitis	342	389	135.8[b]	13.1	16.6	14.0[b]	290	320	68.9[b]	12.4	16.2	12.0[b]
Diarrhoeal diseases	117	123	[c]	4.5	5.2	[c]	108	107	[c]	4.6	5.4	[c]
Maternal mortality	—	—	—	—	—	—	29	24	[c]	1.2	1.2	[c]
Certain diseases of infancy	114	116	[c]	4.4	4.9	[c]	91	90	[c]	3.9	4.6	[c]
Group II	365	543	684.5	14.0	23.1	70.7	372	543	405.2	15.9	27.5	70.4
Neoplasms	36	117	228.9	1.4	5.0	23.6	70	150	148.4	3.0	7.6	25.8
Diseases of circulatory system	329	426	455.6	12.6	18.1	47.1	302	393	256.8	12.9	19.9	44.6
Accidents, suicide, violence	122	108	43.9	4.7	4.6	4.5	40	44	21.6	1.7	2.2	3.8
All other causes	986	706	99.9	37.7	30.1	10.3	848	570	77.5	36.2	28.9	13.4

Sources: For 1861 and 1901, S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972), pp. 240, 242; for 1982, calculated from the World Health Organization data bank.

NOTE: Sums of detail do not in all cases equal totals for all causes, because of rounding.

[a] The population employed for standardization of death rates is the one used by Preston, Keyfitz and Schoen (*op. cit.*, p. 48), namely the Coale-Demeny "West" female stable population, with life expectancy of 65 years and growth rate of 1 per cent.

[b] Includes emphysema and asthma.

[c] Amount negligible; included with "All other causes".

countries, and it implies that the percentages of all deaths attributed to specific causes at these early dates are understated.

With regard to the circulatory diseases, the data for England and Wales in table 15 show that the percentage of all deaths attributable to this group of causes increased from 18 to 47 for males between 1901 and 1982, and from 20 to 45 per cent for females. However, between those dates the age-standardized death rate increased only slightly for males, from 426 to 456 per 100,000 population (an increase of 7 per cent), and declined substantially for females, from 393 to 257 per 100,000, a decline of 35 per cent. As the "All other causes" category in 1901 included a large percentage of deaths from unknown causes, the death rates from diseases of the circulatory system are likely understated, implying that mortality from the cardio-vascular diseases has probably declined for males as well as for females. The increases in the relative importance of deaths from circulatory diseases are therefore due not to increased death rates from those diseases as a group, but on the one hand to more accurate diagnoses in the data for 1982, and on the other hand to the very large declines in mortality from the group I diseases. Declines in circulatory disease mortality were also found by Preston and Nelson in their analysis of data for 165 national populations covering the period 1861 to 1964. When deaths rates from "other and unknown" causes were held constant, declines in circulatory disease mortality accounted for one fourth of the total mortality decline (Preston and Nelson, 1974, p. 29).

Within the group I causes in England and Wales, the infectious and parasitic diseases have declined from 22 per cent (1861) to 0.4 per cent (1982) of total deaths for males, and from 24 per cent to 0.5 per cent for females in the same period. On the other hand, within the group II causes, diseases of the circulatory system have increased relatively from 18 per cent of male deaths (1901) to 47 per cent (1982), and from 20 per cent of female deaths (1901) to 45 per cent (1982). The percentage of deaths from neoplasms increased from 5 to 24 for males between 1901 and 1982, and from 8 to 26 for females.

Accidents, suicide and violence accounted for about 5 per cent of male deaths in 1861, 1901 and 1982, but for females the percentage increased from 2 to 4 per cent between 1861 and 1982.

C. *Contribution of age groups to the widening sex differential in life expectancy, 1900s to 1980s*

In the nineteenth and early twentieth centuries, the age patterns of contributions to the sex differential in life expectancy in the now developed countries were very different from current patterns. This is illustrated by data for England and Wales and Italy in tables 16 and 17, respectively.

In England and Wales, sex differences in infant mortality were by far the largest contributor to the sex differential in life expectancy in the late nineteenth and early twentieth centuries, contributing about 1.5 years (between 40 and 45 per cent) to the total sex differential in 1871-1880 and 1901-1910 (table 16, parts A and C). The only other sizeable contributions during this early period were from ages 35-54, which contributed 0.8 year (between 20 and 24 per cent) at both dates, and ages 55-64, which contributed 0.5 year (13 per cent) in 1901-1910. The combined contribution of the two oldest age groups (65-74 and 75 and over) was only 0.3 year (9 per

TABLE 16. CONTRIBUTION OF AGE GROUPS TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, ENGLAND AND WALES, 1871-1983

Period	All ages	0-1	1-14	15-34	35-54	55-64	65-74	75+
A. *Contribution of age groups to the sex differential in e_0 (years)*								
1871-1880	3.29	1.48	0.22	0.20	0.77	0.34	0.20	0.09
1901-1910	3.87	1.55	0.12	0.37	0.80	0.51	0.35	0.17
1930-1932	4.11	1.12	0.24	0.24	0.82	0.65	0.69	0.34
1950-1954	5.21	0.51	0.15	0.31	0.84	1.38	1.31	0.70
1960-1964	6.00	0.38	0.14	0.46	0.85	1.58	1.65	0.93
1970-1974	6.27	0.33	0.12	0.45	0.87	1.47	1.87	1.17
1980-1983	5.99	0.21	0.08	0.44	0.76	1.29	1.77	1.44
B. *Contribution of age groups to changes in the sex differential in e_0 (years)*[a]								
1901-1910 to 1930-1932	0.25	−0.43	0.12	−0.14	0.03	0.14	0.34	0.18
1930-1932 to 1950-1954	1.09	−0.62	−0.09	0.08	0.02	0.73	0.62	0.36
1950-1954 to 1970-1974	1.07	−0.17	−0.03	0.14	0.02	0.09	0.56	0.47
1970-1974 to 1980-1983	−0.29	−0.13	−0.04	−0.01	−0.10	−0.18	−0.09	0.27
1901-1910 to 1950-1954	1.34	−1.04	0.03	−0.06	0.04	0.87	0.95	0.54
1950-1954 to 1980-1983	0.78	−0.30	−0.07	0.13	−0.08	−0.09	0.47	0.73
1901-1910 to 1980-1983	2.12	−1.34	−0.04	0.07	−0.04	0.78	1.42	1.27
C. *Contribution of age groups to the sex differential in e_0 (percentage)*								
1871-1880	100.0	44.9	6.5	5.9	23.4	10.5	6.0	2.8
1901-1910	100.0	40.0	3.1	9.7	20.6	13.2	9.1	4.3
1930-1932	100.0	27.3	5.9	5.7	20.0	15.9	16.8	8.3
1950-1954	100.0	9.7	2.9	6.0	16.2	26.6	25.1	13.5
1960-1964	100.0	6.4	2.4	7.7	14.2	26.3	27.5	15.5
1970-1974	100.0	5.3	1.9	7.2	13.8	23.4	29.8	18.6
1980-1983	100.0	3.4	1.4	7.3	12.7	21.5	29.6	24.0
D. *Contribution of age groups to changes in the sex differential in e_0 (percentage)*								
1901-1910 to 1930-1932	100.0	−173.9	50.2	−56.3	11.0	58.8	138.4	71.8
1930-1932 to 1950-1954	100.0	−56.5	−8.4	7.0	1.6	66.8	56.3	33.2
1950-1954 to 1970-1974	100.0	−16.3	−3.3	13.1	2.2	8.2	52.5	43.6
1970-1974 to 1980-1983	100.0	44.2	12.6	4.6	36.5	63.2	33.3	−94.4
1901-1910 to 1950-1954	100.0	−78.0	2.3	−4.6	3.3	65.4	71.4	40.3
1950-1954 to 1980-1983	100.0	−38.4	−9.1	16.2	−10.2	−11.9	59.5	93.9
1901-1910 to 1980-1983	100.0	−63.4	−1.9	3.1	−1.7	36.9	67.0	60.1

Sources: Contributions of age groups to the sex differential in life expectancy at birth have been obtained by applying the formulae given in note 2 of the text to life tables for the periods indicated. The life tables for 1871-1880, 1901-1910 and 1930-1932 are among the country life tables that served as a basis for the Coale-Demeny regional model life tables; life tables for the 1950s to the 1980s were calculated by the United Nations from the World Health Organization data bank.

[a] The values shown for each interval are based on the values in Part A of the table, and have been calculated by subtracting the values pertaining to the earlier of the two life tables from the values pertaining to the later life table.

cent) in 1871-1880 and 0.5 year (13 per cent) in 1901-1910.

Parts B and D of table 16 show the absolute and relative contributions of age groups to changes in the sex differential in life expectancy in successive time intervals. Between 1901-1910 and 1980-1983, the sex differential widened from 3.9 years to 6.0 years. During this period, the contribution from infant mortality declined from 1.5 years to only 0.2 year, thus exerting a narrowing effect of 1.3 years on the sex differential. The changing contribution from ages 1-14 also had a slight narrowing effect on the sex differential. The contribution of ages 35-54 remained stable at about 0.8 year throughout the century (part A). In all the remaining age groups, the absolute contributions increased between 1901-1910 and 1980-1983: from 0.2 to 0.4 year at ages 15-34; from 0.3 to 1.3 years (ages 55-64); from 0.2 to 1.8 years (ages 65-74); and from 0.1 to 1.4 years (ages 75 and over). These data show that the widening of the sex differential was due almost entirely to ages 55-64, 65-74 and 75 and over, which contributed 0.8 year (37 per cent), 1.4 years (67 per cent) and 1.3 years (60 per cent), respectively, to the increase in the sex differential in life expectancy (parts B and D). That the sum of these three percentages exceeds 100 per cent reflects, of course, the fact that some age groups made negative contributions.

It was mentioned earlier that England and Wales was the first of several countries to experience a narrowing of the sex differential in life expectancy in recent years. As shown by the data for the interval 1970-1974 to 1980-1983 (table 16, part B), all age groups but the oldest contributed to the narrowing of the differential from 6.3 to 6.0 years.

In Italy (table 17), the overall increase in the sex differential from 0.5 year in 1881-1882 to 6.7 years in 1980 was far greater than in England and Wales. In 1881-1882 and 1899-1902, several age groups (particu-

TABLE 17. CONTRIBUTION OF AGE GROUPS TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, ITALY, 1881-1980

Period	All ages	0-1	1-14	15-34	35-54	55-64	65-74	75+
A. *Contribution of age groups to the sex differential in e_0 (years)*								
1881-1882	0.50	0.92	−0.20	−0.32	0.14	0.07	−0.08	−0.04
1899-1902	0.34	0.85	−0.44	−0.38	0.16	0.17	0.01	−0.03
1921	1.53	0.81	0.12	0.03	0.24	0.24	0.07	0.02
1931	2.08	0.73	0.00	0.08	0.55	0.36	0.24	0.12
1950-1954	3.69	0.62	0.13	0.38	0.88	0.82	0.52	0.34
1960-1964	5.34	0.55	0.14	0.63	1.06	1.34	1.09	0.55
1970-1974	6.16	0.44	0.14	0.57	1.19	1.38	1.58	0.87
1980	6.68	0.30	0.08	0.61	1.26	1.60	1.70	1.13
B. *Contribution of age groups to changes in the sex differential in e_0 (years)*[a]								
1899-1902 to 1931	1.74	−0.12	0.44	0.46	0.40	0.19	0.23	0.14
1931 to 1950-1954	1.61	−0.11	0.13	0.30	0.32	0.46	0.28	0.22
1950-1954 to 1970-1974	2.48	−0.17	0.01	0.19	0.32	0.55	1.05	0.53
1970-1974 to 1980	0.52	−0.14	−0.06	0.05	0.07	0.22	0.12	0.26
1899-1902 to 1950-1954	3.35	−0.23	0.57	0.76	0.72	0.65	0.51	0.36
1950-1954 to 1980	2.99	−0.32	−0.05	0.24	0.39	0.77	1.17	0.79
1899-1902 to 1980	6.34	−0.55	0.52	0.99	1.11	1.42	1.69	1.15
C. *Contribution of age groups to the sex differential in e_0 (percentage)*								
1881-1882	100.00	184.03	−39.72	−64.27	27.15	14.77	−14.97	−6.99
1899-1902	100.00	250.74	−129.79	−111.80	45.72	50.15	2.65	−7.67
1921	100.00	53.28	7.73	2.03	15.47	15.99	4.46	1.05
1931	100.00	35.26	−0.14	3.66	26.54	17.39	11.75	5.54
1950-1954	100.00	16.78	3.55	10.25	23.75	22.31	14.20	9.16
1960-1964	100.00	10.22	2.68	11.77	19.77	24.99	20.37	10.20
1970-1974	100.00	7.20	2.35	9.18	19.33	22.30	25.56	14.06
1980	100.00	4.50	1.26	9.19	18.93	23.87	25.37	16.88
D. *Contribution of age groups to changes in the sex differential in e_0 (percentage)*								
1899-1902 to 1931	100.00	−6.79	25.16	26.19	22.80	11.00	13.53	8.12
1931 to 1950-1954	100.00	−7.01	8.31	18.72	20.15	28.64	17.36	13.83
1950-1954 to 1970-1974	100.00	−7.07	0.57	7.59	12.76	22.29	42.49	21.37
1970-1974 to 1980	100.00	−27.66	−11.80	9.28	14.12	42.55	23.02	50.48
1899-1902 to 1950-1954	100.00	−6.90	17.04	22.60	21.52	19.49	15.37	10.87
1950-1954 to 1980	100.00	−10.62	−1.57	7.89	13.00	25.79	39.12	26.39
1899-1902 to 1980	100.00	−8.66	8.26	15.66	17.50	22.47	26.58	18.19

Sources: Contributions of age groups to the sex differential in life expectancy at birth have been obtained by applying the formulae given in note 2 of the text to life tables for the periods indicated. The life tables for 1881-1882 and 1899-1902 are among the country life tables that served as a basis for the Coale-Demeny regional model life tables; life tables for 1921 and 1931 are from S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972); life tables for the 1950s to 1980 were calculated by the United Nations from the World Health Organization data bank.

[a] See table 16, note a.

larly 1-14 and 15-34) made a negative contribution to the sex differential, reflecting higher female mortality compared with male mortality at those ages. Higher female mortality during childhood, adolescence and the female reproductive ages was not uncommon in Europe in the nineteenth and early twentieth centuries (Tabutin, 1978; Vidal, 1980). In Italy, the contribution from infant mortality also declined between 1899-1902 and 1980, resulting in a negative contribution to the sex differential, but by a much smaller amount than in England and Wales (−0.6 year in Italy compared with −1.3 years in England and Wales). Other differences between the two countries occurred at ages 15-34 and 35-54. These two age groups made virtually no contribution to increases in the sex differential in life expectancy in England and Wales, but contributed about one year each in Italy. As in England and Wales, the three oldest age groups contributed the most to increases in the sex differential in life expectancy in Italy between 1899-1902 and 1980: ages 55-64, 1.4 years (23 per cent); ages 65-74, 1.7 years (27 per cent); ages 75 and over, 1.2 years (18 per cent).

The different trends observed in Italy compared with England and Wales are due in part to the fact that England and Wales were at a more advanced stage of the demographic transition at the turn of the century (life expectancy at birth averaged four years less in Italy), but in the early 1980s life expectancy was similar in both countries.

The patterns of age contributions to changes in the sex differential in life expectancy are summarized in table 18 for 14 developed countries. In all of the countries, trends in infant mortality (age 0) had a negative effect on the sex differential. The mean contribution from this age

TABLE 18. CONTRIBUTION OF AGE GROUPS TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY AT BIRTH, SELECTED DEVELOPED COUNTRIES, 1900s TO 1980s

Region, country and period	All ages		Contribution of age group (per cent)						
	Years	Per cent	0	1-14	15-34	35-54	55-64	65-74	75+
Northern America									
United States									
1900-1902 to 1950-1954	3.02	100.0	−25.3	−5.0	20.8	27.4	37.0	30.5	14.6
1950-1954 to 1980-1982	1.66	100.0	−20.4	−4.5	16.9	−2.0	2.7	38.5	68.9
1900-1902 to 1980-1982	4.68	100.0	−23.6	−4.8	19.4	16.9	24.8	33.3	33.9
East Asia									
Japan									
1899 to 1950-1954	2.30	100.0	−23.7	8.1	45.6	16.4	15.1	24.8	13.7
1950-1954 to 1980-1983	2.11	100.0	−15.0	1.3	9.3	20.1	10.2	27.0	47.2
1899 to 1980-1983	4.41	100.0	−19.6	4.8	28.2	18.1	12.8	25.9	29.8
Europe									
Eastern Europe									
Czechoslovakia									
1899-1902 to 1955-1959	2.22	100.0	−66.6	20.1	38.0	8.7	46.7	41.7	11.3
1955-1959 to 1980-1982	2.31	100.0	−6.1	−2.8	0.7	41.1	19.1	24.9	22.9
1899-1902 to 1980-1982	4.53	100.0	−35.7	8.4	19.0	25.3	32.6	33.2	17.2
Northern Europe									
Denmark									
1901-1905 to 1950-1954	−0.63	100.0	164.4	−35.2	−81.6	43.7	1.4	2.5	4.8
1950-1954 to 1980-1983	3.51	100.0	−12.8	−3.8	6.0	12.0	20.3	39.0	39.2
1901-1905 to 1980-1983	2.88	100.0	−51.5	3.1	25.2	5.1	24.5	46.9	46.7
Finland									
1901-1905 to 1950-1954	3.61	100.0	−17.8	6.8	21.2	35.6	28.9	20.6	4.8
1950-1954 to 1980-1983	2.09	100.0	−21.7	−8.6	3.0	4.9	22.9	49.2	50.3
1901-1904 to 1980-1983	5.70	100.0	−19.2	1.1	14.5	24.3	26.7	31.1	21.4
Sweden									
1901-1910 to 1950-1954	0.39	100.0	−154.7	51.7	96.9	11.5	39.6	40.7	14.3
1950-1954 to 1980-1983	3.32	100.0	−9.7	−4.6	0.9	16.1	20.2	36.6	40.6
1901-1910 to 1980-1983	3.71	100.0	−25.0	1.3	11.0	15.6	22.2	37.0	37.8
United Kingdom									
England and Wales									
1901-1910 to 1950-1954	1.34	100.0	−78.0	2.3	−4.6	3.3	65.5	71.5	39.9
1950-1954 to 1980-1983	0.78	100.0	−38.3	−9.1	16.2	−10.2	−12.1	59.1	94.4
1901-1910 to 1980-1983	2.12	100.0	−63.3	−1.9	3.1	−1.7	36.8	66.9	60.0
Southern Europe									
Italy									
1899-1902 to 1950-1954	3.35	100.0	−6.9	17.0	22.6	21.5	19.5	15.4	10.9
1950-1954 to 1980	2.99	100.0	−10.6	−1.6	7.9	13.0	25.8	39.1	26.4
1899-1902 to 1980	6.34	100.0	−8.7	8.3	15.7	17.5	22.5	26.6	18.2
Spain									
1900 to 1950	2.96	100.0	−5.3	4.3	11.7	23.9	25.9	23.2	16.3
1950 to 1975-1979	1.15	100.0	−34.7	0.7	−2.9	5.0	27.8	67.7	36.4
1900 to 1975-1979	4.11	100.0	−13.5	3.3	7.6	18.7	26.4	35.6	21.9
Western Europe									
Austria									
1900-1904 to 1950-1954	3.13	100.0	−31.6	13.7	27.4	19.0	32.4	28.5	10.5
1950-1954 to 1980-1983	1.91	100.0	−37.5	−4.9	20.8	33.1	16.0	38.5	33.9
1900-1904 to 1980-1983	5.04	100.0	−33.8	6.7	24.9	24.3	26.2	32.3	19.4
Belgium									
1891-1900 to 1950-1954	1.56	100.0	−47.1	9.8	24.7	14.0	42.4	34.1	22.1
1950-1954 to 1980-1981	1.82	100.0	−27.3	−8.8	17.8	−2.2	20.2	58.0	42.3
1891-1900 to 1980-1981	3.37	100.0	−36.5	−0.2	21.0	5.3	30.4	47.0	33.0
France									
1901-1910 to 1950-1954	2.51	100.0	−28.7	5.0	13.8	18.5	34.5	34.1	22.7
1950-1954 to 1980-1983	2.37	100.0	−22.8	−2.4	16.9	16.8	18.4	34.2	38.8
1901-1910 to 1980-1983	4.88	100.0	−25.8	1.4	15.3	17.7	26.7	34.2	30.5
Netherlands									
1900-1909 to 1950-1954	0.03	100.0	−3525.9	148.1	940.7	711.1	829.6	577.8	418.5
1950-1954 to 1980-1983	4.36	100.0	−5.9	−2.9	1.4	9.1	21.1	40.5	36.7
1900-1909 to 1980-1983	4.38	100.0	−27.6	−2.0	7.2	13.4	26.1	43.8	39.1

TABLE 18 (*cont.*)

Region, country and period	All ages		Contribution of age group (per cent)						
	Years	Per cent	0	1-14	15-34	35-54	55-64	65-74	75+
Oceania									
Australia-New Zealand									
Australia									
1901-1910 to 1950-1954.....	1.84	100.0	−31.5	1.0	34.3	0.5	38.0	39.8	17.9
1950-1954 to 1980-1982.....	1.66	100.0	−12.1	−5.6	4.9	12.5	5.1	36.8	58.4
1901-1910 to 1980-1982.....	3.49	100.0	−22.3	−2.1	20.4	6.2	22.4	38.4	37.1
Means, 14 countries (years)									
1900s to 1950s	1.97		−0.8	0.2	0.6	0.4	0.7	0.6	0.3
1950s to 1980s	2.29		−0.4	−0.1	0.2	0.3	0.4	0.9	0.9
1900s to 1980s	4.26		−1.1	0.1	0.7	0.7	1.1	1.5	1.3
Means, 14 countries (per cent)[a]									
1900s to 1950s		100.0	−38.3	9.6	27.9	19.6	34.3	31.3	15.5
1950s to 1980s		100.0	−16.4	−3.8	7.5	13.6	17.7	39.9	41.5
1900s to 1980s		100.0	−26.4	2.4	16.9	16.4	25.3	35.9	29.4

Sources: The results presented here have been obtained by applying the same methodology described in table 16, sources and note a, to life tables for the periods indicated. The life tables for periods prior to 1950 are among the country life tables that served as a basis for the Coale-Demeny regional model life tables. Life tables for the 1950s to the 1980s were calculated by the United Nations from the World Health Organization data bank.

[a] Means were calculated by first averaging the absolute contributions (i.e., in years) of age groups to the sex differential for the 14 countries, then obtaining a percentage distribution based on those averages.

group to changes in the sex differential between 1900 and the 1980s was −1.1 years. At ages 1-14, contributions were generally small, whether negative as in the case of Australia, Belgium, England and Wales, the Netherlands and the United States, or positive, as in all the other countries. Ages 15-34 and 35-54 contributed, on average, 0.7 year each. In most countries, the three oldest age groups were the largest contributors to the increasing sex differential in life expectancy. The means, as shown in table 18, were: ages 55-64, 1.1 years; ages 65-74, 1.5 years; and ages 75 and over, 1.3 years.

The data for the two intervals, 1900-1950s and 1950s-1980s, show that in the first interval, the age groups making the greatest positive contribution to the changing sex differential were 15-34, 55-64 and 65-74, each age group contributing 0.6 or 0.7 year. Between the 1950s and the 1980s, the two oldest age groups (65-74 and 75 and over) were the largest contributors (0.9 year each).

D. *Contribution of causes of death to the widening sex differential in life expectancy, 1900s to 1980s*

In this section, the contributions of causes of death to changes in the sex differential in life expectancy between the 1900s and the 1980s are considered. The analysis is based on a decomposition of the sex differential in life expectancy at birth by the method described in note 3. The causes of death selected for the long-term analysis are, with a few exceptions, those for which age-specific death rates are provided in Preston, Keyfitz and Schoen (1972). The exceptions include several causes for which mortality is now extremely low in developed countries, namely, respiratory tuberculosis, maternal mortality, certain diseases of infancy and diarrhoeal disease. Those causes have not been tabulated for the 1980s. On the other hand, more detailed causes than were available from Preston, Keyfitz and Schoen have been tabulated for diseases of the circulatory system and neoplasms for the 1950s, 1970 and the 1980s.

Two sets of tabulations were prepared. In the first, the contributions of causes of death to the changing sex differentials in life expectancy between around 1900 and the early 1980s were calculated for five countries selected for geographic diversity (Australia, England and Wales, Italy, Sweden and the United States). The results are presented in tables 19 to 23. In the second, the contributions of causes of death were calculated for 23 countries for the period covered by the World Health Organization data bank, that is, from the early 1950s to the present (table 24).

1. *Diseases of the circulatory system*

Diseases of the circulatory system have been by far the largest contributor to the widening of the sex differential in life expectancy. Around 1900 this group of causes made a relatively small absolute contribution to the sex differential. In the five countries in tables 19 to 23, the contribution ranged from −0.2 year in Italy to 0.4 year in Australia, and averaged 0.1 year. By the early 1980s, contributions from the circulatory diseases ranged from 2.1 years in Italy to 3.3 years in Sweden, with an average of 2.9 years. Figure VI, part *e*, shows how sex ratios of death rates for the circulatory diseases have increased in England and Wales between 1901 and 1982.

The most important component of circulatory disease mortality currently in most developed countries is ischaemic heart disease. Contributions from this component in the five countries are shown separately for the 1950s to the 1980s in tables 19 to 23, and trends in sex ratios of death rates for ischaemic heart disease are illustrated for England and Wales in figure VI, part *f*. During this period, ischaemic heart disease was responsible for a

large part of the increase in the sex differential in life expectancy attributable to diseases of the circulatory system in four of the five countries. The other component of circulatory diseases shown separately is cerebro-vascular disease. Although it is a major cause of death, the differences between male and female death rates from this cause are relatively small, and it therefore makes only a small contribution to the sex differential. In the five countries, cerebro-vascular disease increased its contribution slightly between the 1950s and the 1980s, but only by a maximum of about 0.3 year in Australia (from −0.15 to 0.15) and Sweden (from −0.14 to 0.13).

The averages for 23 developed countries (table 24) show an increase of 1.6 years (from 1.2 to 2.8 years) in the contribution of diseases of the circulatory system to the sex differential in life expectancy between the 1950s and the early 1980s. Ischaemic heart disease accounted for 1.0 year of this increase and cerebro-vascular disease for 0.3 year, with other circulatory diseases accounting for the remaining 0.3 year increase.

The large increase in the contribution of the circulatory diseases to the sex differential in life expectancy reflects in part changes in the composition of this category. Mortality from some of the circulatory diseases that affected both sexes nearly equally, or where female mortality was higher than that of males, has decreased. For example, mortality from rheumatic heart disease, which is infectious in origin, and for which female mortality had generally been equal to or higher than that of males, has declined greatly along with mortality from other infectious diseases. Mortality from cerebro-vascular disease, which affected both sexes nearly equally, has also declined. On the other hand, mortality from coronary artery disease (ischaemic heart disease), which displays a greater masculinity of mortality than other components of the circulatory diseases, has increased.

In addition to the changes in the composition of the circulatory diseases, there has been a general tendency for female mortality trends to be more favourable than those of males for all components of the circulatory diseases. (Some of these trends can be seen in data for the United States in Moriyama, Krueger and Stamler, 1971, pp. 441-446; and in United States, 1985, pp. 46-51).

2. *Neoplasms*

Malignant neoplasms (cancer) are a group of diseases with diverse aetiology that have in common a pathological proliferation of cells. Together they have made the second largest contribution, after diseases of the circulatory system, to the widening of the sex differential in life expectancy between 1900 and the 1980s.

Data for the five countries in tables 19 to 23 show that around 1900, neoplasms made a negative contribution to the sex differential in four of the countries, reflecting higher female than male mortality. The contributions ranged from 0 (Sweden, 1911) to −0.5 year (United States, 1900), with an average of −0.2 year in the five countries. In the early 1930s, negative contributions are still observed for the United States (−0.4 year), Italy (−0.1 year) and Sweden (−0.1 year), with an average of −0.1 year. Between the early 1930s and 1951, increases of 0.5 or 0.6 year in the contribution are observed in England and Wales (from 0 to 0.6 year) and the United States (from −0.4 to 0.1 year), with smaller increases in the other three countries. The mean contribution for the five countries in 1951 was 0.2 year.

According to the figures given in the previous paragraph, the increase in the contribution of neoplasms to the sex differential in life expectancy between 1900 and 1951 was slight, only 0.4 year on average. Much larger increases, averaging 0.9 year in the five countries, occurred between 1951 and the early 1980s. During that period, the contribution increased by 1.4 years in Italy, by 1.1 years in Australia and the United States and by 0.7 year in Sweden. In England and Wales, which had already experienced an increase of nearly a year between 1901 and 1951, the increase between 1951 and 1982 was only 0.4 year. The mean contribution from neoplasms in the five countries in the early 1980s was 1.2 years, and ranged from 0.7 year in Sweden (1984) to 1.7 years in Italy (1980).

Means for the 23 countries in table 24 show a trend similar to that of the five countries. The contribution of neoplasms to the sex differential in life expectancy increased by 1.0 year, from 0.3 year in the early 1950s to 1.3 years in the early 1980s.

The negative contributions from neoplasms observed in the data for the early decades of the twentieth century reflect the fact that female mortality from neoplasms was generally much higher than that of males over a broad range of ages beginning at 20-24 or 25-29 years. Although the female disadvantage in these age groups has decreased gradually with time, in the early 1980s female mortality from neoplasms was still higher than that of males in most developed countries in certain age groups, most commonly ages 30-50 years. The cancers responsible for female excess mortality at these ages are cancer of the breast and female genital organs.

The graph of age-specific sex ratios of death rates from neoplasms in England and Wales (figure VI, part *c*) illustrates the trend towards increasing sex ratios of death rates. In 1901, female mortality from neoplasms in England and Wales was higher than that of males in all age groups above 20 years (i.e., the sex ratios were below unity). From ages 25-29 to 50-54, the sex ratio was below 0.7, and at ages 35-39, it was only 0.4 (conversely, female mortality from neoplasms was 2.4 times higher than that of males). The graph shows large increases with time in sex ratios of death rates, particularly at ages 70-74 to 80-84, where ratios increased from 0.8 or 0.9 in 1901 to over 2.0 in 1982. However, the ratios, which had increased at all ages between 1901 and 1951, began to decrease again in some age groups after 1951, and in other age groups after 1970, with the result that sex ratios for neoplasms in 1983 were substantially below those of 1951 for ages 20-60. The decreases observed in the ratios for neoplasms since 1951 are mainly due to less favourable mortality trends for lung cancer among females below age 65. The striking changes in patterns of sex ratios of death rates for lung

Figure VI. Trends in sex ratios of death rates by cause, England and Wales, 1901-1982

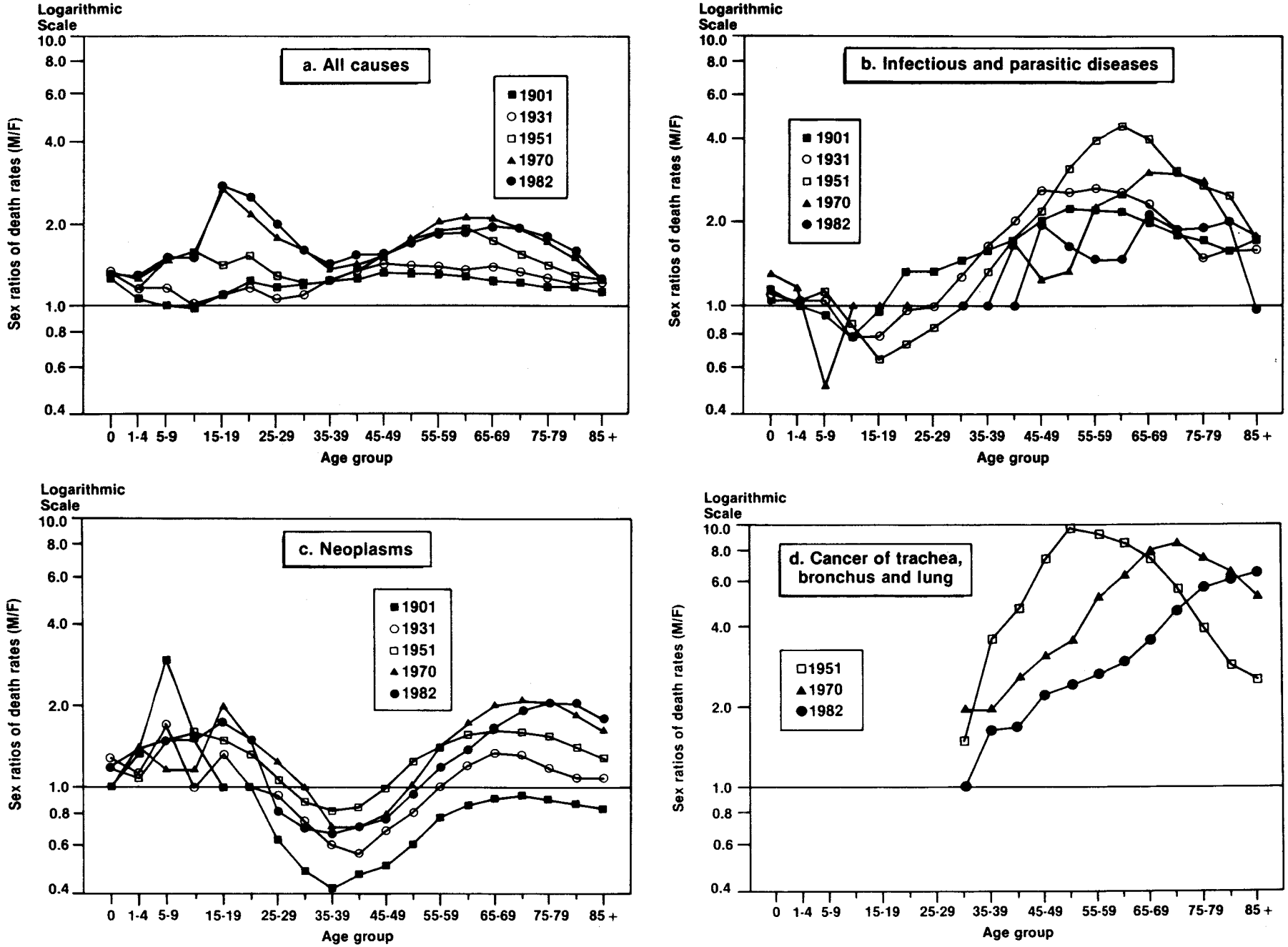
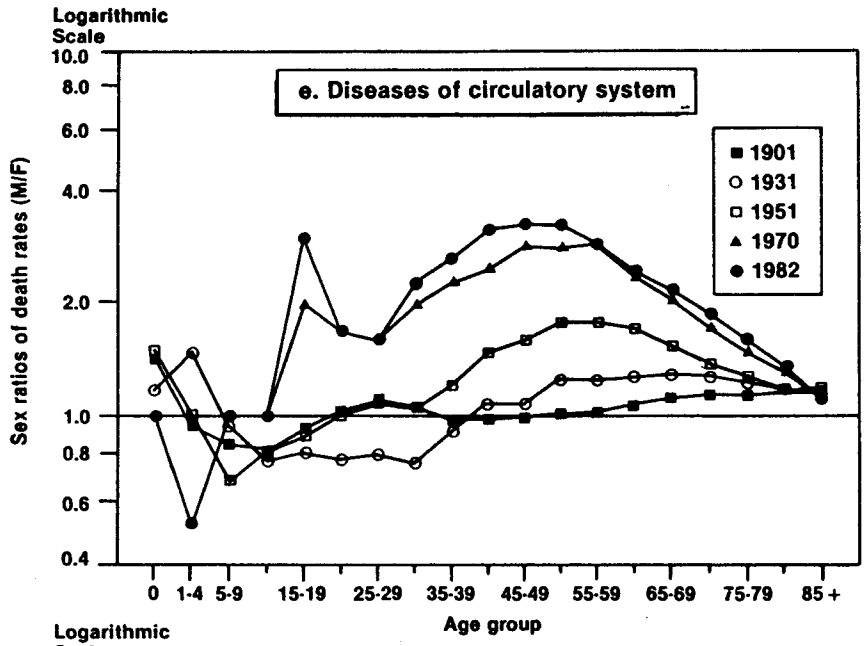
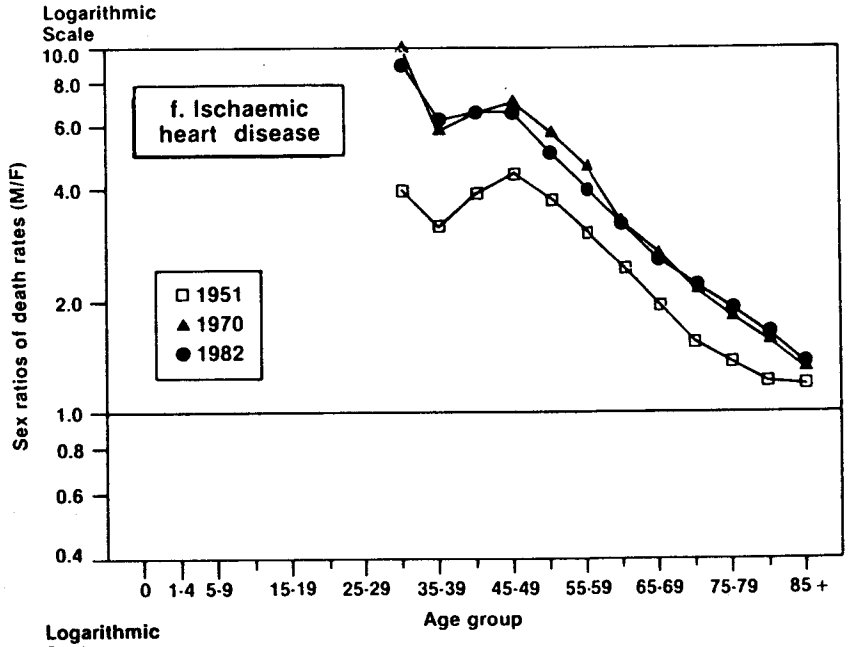
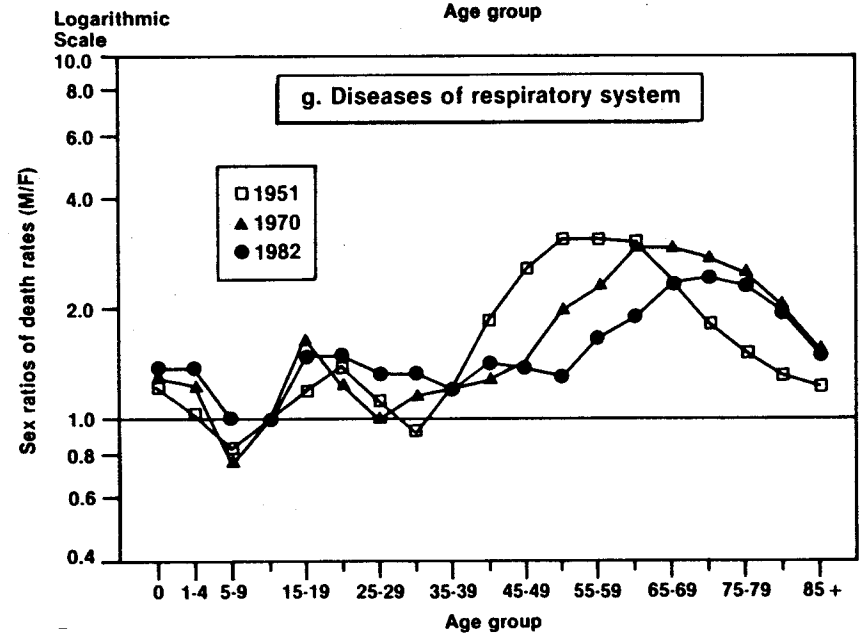
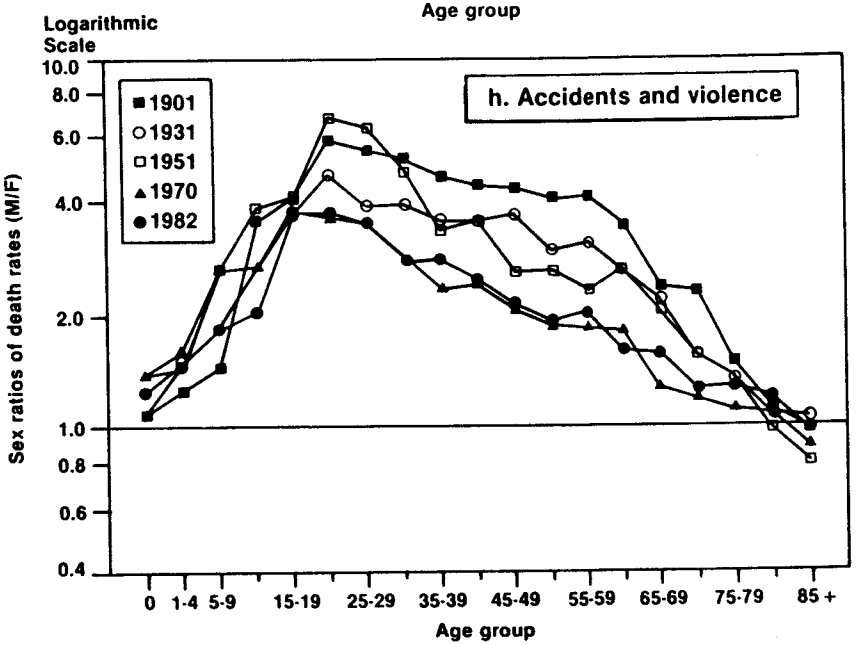


Figure VI (*cont.*)









Sources: For 1901 and 1931, S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death; Life Tables for National Populations* (New York and London, Seminar Press, 1972). For 1951, 1970 and 1982, World Health Organization data bank.

TABLE 19. CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AUSTRALIA, 1911-1983

Cause of death	Contribution of causes to the sex differential in life expectancy (years)					Contribution of causes to changes in the sex differential in life expectancy: Years[a]			Per cent		
	1911 (1)	1933 (2)	1951 (3)	1970 (4)	1983 (5)	1911 to 1951	1951 to 1983	1911 to 1983	1911 to 1951	1951 to 1983	1911 to 1983
All causes	3.83	3.87	5.48	6.78	6.81	1.65	1.33	2.98	100.0	100.0	100.0
I. Infectious and parasitic diseases; pneumonia, influenza, bronchitis;[b] diarrhoeal disease; certain diseases of infancy	1.40	1.19	0.85	1.13	0.74	−0.55	−0.11	−0.66	−33.3	−8.3	−22.1
II. Maternal mortality	−0.58	−0.12	−0.13	−0.03	0.00	0.45	0.13	0.58	27.3	9.8	19.5
III. Neoplasms[c,d]	−0.09	0.10	0.22	0.73	1.27	0.31	1.05	1.36	18.8	78.9	45.6
Trachea, bronchus, lung[e]	. .	. .	0.19	0.55	0.77	[f]	0.58	[f]	[f]	43.6	[f]
Female breast[e]	. .	. .	−0.31	−0.35	−0.46	[f]	−0.15	[f]	[f]	−11.3	[f]
IV. Diseases of circulatory system[d]	0.36	0.75	2.10	3.09	2.91	1.74	0.81	2.55	105.5	60.9	85.6
Ischaemic heart disease	. .	. .	1.92	2.72	2.39	[f]	0.47	[f]	[f]	35.3	[f]
Cerebro-vascular disease	. .	. .	−0.15	0.08	0.15	[f]	0.30	[f]	[f]	22.6	[f]
V. Accidents, suicide, violence	1.54	0.93	1.68	1.44	1.22	0.14	−0.46	−0.32	8.5	−34.6	−10.7
VI. All other causes	1.20	1.02	0.76	0.42	0.67	−0.44	−0.09	−0.53	−26.7	−6.8	−17.8

Sources: Contributions of causes of death to the sex differential in life expectancy at birth have been obtained by applying the formulae given in note 3 of the text to life tables for the periods indicated. The life tables for periods prior to 1950 are from S. H. Preston, N. Keyfitz and R. Schoen, *Causes of Death: Life Tables for National Populations* (New York and London, Seminar Press, 1972). Life tables for the 1950s to the 1980s were calculated from the World Health Organization data bank.

. . Not tabulated separately.

[a] Based on the values in columns (1) through (5). Calculated by subtracting the values for the earlier date from those for the later date.

[b] Data for the 1970s and the 1980s also include emphysema and asthma.

[c] Includes benign neoplasms.

[d] The sums of the details for the sub-categories do not add to the totals shown because the sub-categories are not exhaustive.

[e] Malignant neoplasms only.

[f] Not computed because category was not tabulated separately for one or both dates being compared.

TABLE 20. CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, ENGLAND AND WALES, 1901-1982

Cause of death	Contribution of causes to the sex differential in life expectancy (years)					Contribution of causes to changes in the sex differential in life expectancy: Years[a]			Per cent		
	1901 (1)	1931 (2)	1951 (3)	1970 (4)	1982 (5)	1901 to 1951	1951 to 1982	1901 to 1982	1901 to 1951	1951 to 1982	1901 to 1982
All causes	3.98	4.15	5.13	6.32	5.94	1.15	0.81	1.96	100.0	100.0	100.0
I. Infectious and parasitic diseases; pneumonia, influenza, bronchitis;[b] diarrhoeal disease; certain diseases of infancy	2.77	2.20	1.66	1.36	0.86	−1.11	−0.80	−1.91	−96.5	−98.8	−97.4
II. Maternal mortality	−0.39	−0.26	−0.06	−0.02	0.00	0.33	0.06	0.39	28.7	7.4	19.9
III. Neoplasms[c,d]	−0.32	0.01	0.60	1.02	0.96	0.92	0.36	1.28	80.0	44.4	65.3
Trachea, bronchus, lung[e]	. .	. .	0.55	1.00	0.87	[f]	0.32	[f]	[f]	39.5	[f]
Female breast[e]	. .	. .	−0.36	−0.49	−0.59	[f]	−0.23	[f]	[f]	−28.4	[f]
IV. Diseases of circulatory system[d]	0.10	0.55	1.58	2.96	3.04	1.48	1.46	2.94	128.7	180.2	150.0
Ischaemic heart disease	. .	. .	1.37	2.45	2.60	[f]	1.23	[f]	[f]	151.9	[f]
Cerebro-vascular disease	. .	. .	0.08	0.20	0.15	[f]	0.07	[f]	[f]	8.6	[f]
V. Accidents, suicide, violence	0.79	0.79	0.74	0.66	0.63	−0.05	−0.11	−0.16	−4.3	−13.6	−8.2
VI. All other causes	1.03	0.86	0.61	0.34	0.45	−0.42	−0.16	−0.58	−36.5	−19.8	−29.6

Sources and notes: See table 19.

TABLE 21. CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, ITALY, 1901-1980

Cause of death	Contribution of causes to the sex differential in life expectancy (years)					Contribution of causes to changes in the sex differential in life expectancy: Years[a]			Contribution of causes to changes in the sex differential in life expectancy: Per cent		
	1901 (1)	1931 (2)	1951 (3)	1970 (4)	1980 (5)	1901 to 1951	1951 to 1980	1901 to 1980	1901 to 1951	1951 to 1980	1901 to 1980
All causes	0.55	2.08	3.59	6.02	6.68	3.04	3.09	6.13	100.0	100.0	100.0
I. Infectious and parasitic diseases; pneumonia, influenza, bronchitis;[b] diarrhoeal disease; certain diseases of infancy	0.4	1.04	1.07	1.08	0.60	0.62	−0.47	0.15	20.4	−15.2	2.4
II. Maternal mortality	−0.27	−0.25	−0.13	−0.05	0.00	0.14	0.13	0.27	4.6	4.2	4.4
III. Neoplasms[c,d]	−0.13	−0.10	0.24	1.12	1.66	0.37	1.42	1.79	12.2	46.0	29.2
Trachea, bronchus, lung[e]	. .	. .	0.12	0.58	0.95	f	0.83	f	f	26.9	f
Female breast[e]	. .	. .	−0.20	−0.34	−0.40	f	−0.20	f	f	−6.5	f
IV. Diseases of circulatory system[d]	−0.19	0.01	0.45	1.68	2.14	0.64	1.69	2.33	21.1	54.7	38.0
Ischaemic heart disease	. .	. .	0.16	1.00	1.24	f	1.08	f	f	35.0	f
Cerebro-vascular disease	. .	. .	0.25	0.40	0.39	f	0.14	f	f	4.5	f
V. Accidents, suicide, violence	0.46	0.70	0.95	1.19	1.02	0.49	0.07	0.56	16.1	2.3	9.1
VI. All other causes	0.23	0.68	1.01	1.00	1.26	0.78	0.25	1.03	25.7	8.1	16.8

Sources and notes: See table 19.

TABLE 22. CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, SWEDEN, 1911-1984

Cause of death	Contribution of causes to the sex differential in life expectancy (years)					Contribution of causes to changes in the sex differential in life expectancy: Years[a]			Contribution of causes to changes in the sex differential in life expectancy: Per cent		
	1911 (1)	1930 (2)	1951 (3)	1970 (4)	1984 (5)	1911 to 1951	1951 to 1984	1911 to 1984	1911 to 1951	1951 to 1984	1911 to 1984
All causes	2.91	2.04	2.69	5.06	6.24	−0.22	3.55	3.33	100.0	100.0	100.0
I. Infectious and parasitic diseases; pneumonia, influenza, bronchitis;[b] diarrhoeal disease; certain diseases of infancy	0.77	0.46	0.48	0.44	0.40	−0.29	−0.08	−0.37	131.8	−2.3	−11.1
II. Maternal mortality	−0.29	−0.23	−0.07	−0.01	0.00	0.22	0.07	0.29	−100.0	2.0	8.7
III. Neoplasms[c,d]	0.04	−0.10	0.05	0.37	0.73	0.01	0.68	0.69	−4.5	19.2	20.7
Trachea, bronchus, lung[e]	. .	. .	0.09	0.29	0.37	f	0.28	f	f	7.9	f
Female breast[e]	. .	. .	−0.28	−0.39	−0.43	f	−0.15	f	f	−4.2	f
IV. Diseases of circulatory system[d]	0.08	0.16	0.60	2.45	3.25	0.52	2.65	3.17	−236.4	74.6	95.2
Ischaemic heart disease	. .	. .	0.69	2.14	2.78	f	2.09	f	f	58.9	f
Cerebro-vascular disease	. .	. .	−0.14	0.10	0.13	f	0.27	f	f	7.6	f
V. Accidents, suicide, violence	1.58	1.24	1.18	1.20	1.14	−0.40	−0.04	−0.44	181.8	−1.1	−13.2
VI. All other causes	0.73	0.51	0.45	0.61	0.72	−0.28	0.27	−0.01	127.3	7.6	−0.3

Sources and notes: See table 19.

cancer are depicted in part *d* of figure VI. From a sex ratio of nearly 10 at ages 50-54 in 1951, the ratio declined to 2.5 in 1982. This is a particularly marked manifestation of a pattern that is fairly general in developed countries.

The data base for the present study does not permit an analysis of the contributions of neoplasms by site to the widening of the sex differential in life expectancy before the 1950s. However, it is clear that by mid-century, cancer of the respiratory system was increasing rapidly among adult males in developed countries. In the United States, the age-standardized death rate from cancer of the bronchus and lung among white males increased nearly tenfold from 3.9 to 35.3 per 100,000 between 1930 and 1959-1961 (Lilienfeld, Levin and Kessler, 1972, p. 17). The corresponding rates for white females were 2.3 (1930) and 5.2 (1959-1961).

The contributions of selected cancer sites to changes in the sex differential in life expectancy between the early 1950s and the early 1980s are shown in table 24. For

TABLE 23. CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, UNITED STATES, 1900-1982

Cause of death	Contribution of causes to the sex differential in life expectancy (years)					Contribution of causes to changes in the sex differential in life expectancy: Years[a]			Contribution of causes to changes in the sex differential in life expectancy: Per cent		
	1900 (1)	1930 (2)	1951 (3)	1970 (4)	1982 (5)	1900 to 1951	1951 to 1982	1900 to 1982	1900 to 1951	1951 to 1982	1900 to 1982
All causes	2.61	3.44	5.73	7.50	7.45	3.12	1.72	4.84	100.0	100.0	100.0
I. Infectious and parasitic diseases; pneumonia, influenza, bronchitis;[b] diarrhoeal disease; certain diseases of infancy	1.38	1.43	0.87	0.80	0.33	−0.51	−0.54	−1.05	−16.3	−31.4	−21.7
II. Maternal mortality	−0.34	−0.52	−0.10	−0.02	0.00	0.24	0.10	0.34	7.7	5.8	7.0
III. Neoplasms[c,d]	−0.45	−0.43	0.10	0.79	1.20	0.55	1.10	1.65	17.6	64.0	34.1
Trachea, bronchus, lung[e]	. .	. .	0.25	0.65	0.80	[f]	0.55	[f]	[f]	32.0	[f]
Female breast[e]	. .	. .	−0.36	−0.42	−0.50	[f]	−0.14	[f]	[f]	−8.1	[f]
IV. Diseases of circulatory system[d]	0.14	0.65	2.58	3.36	3.18	2.44	0.60	3.04	78.2	34.9	62.8
Ischaemic heart disease	. .	. .	2.29	2.83	2.34	[f]	0.05	[f]	[f]	2.9	[f]
Cerebro-vascular disease	. .	. .	0.08	0.18	0.13	[f]	0.05	[f]	[f]	2.9	[f]
V. Accidents, suicide, violence	1.23	1.80	1.62	1.83	1.65	0.39	0.03	0.42	12.5	1.7	8.7
VI. All other causes	0.65	0.51	0.66	0.74	1.09	0.01	0.43	0.44	0.3	25.0	9.1

Sources and notes: See table 19.

TABLE 24. AVERAGE CONTRIBUTION OF CAUSES OF DEATH TO THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, AND TO CHANGES IN THE SEX DIFFERENTIAL IN LIFE EXPECTANCY, 23 DEVELOPED COUNTRIES, 1950s, 1970, 1980s

Cause of death	Contribution of causes to the sex differential in life expectancy (years)			Contribution of causes to changes in the sex differential in life expectancy: 1950s to 1970		1970 to 1980s		1950s to 1980s	
	1950s	1970	1980s	Years	Per cent	Years	Per cent	Years	Per cent
All causes	4.52	6.35	6.91	1.83	100.0	0.55	100.0	2.38	100.0
Infectious and parasitic diseases	0.39	0.16	0.06	−0.23	−12.7	−0.10	−17.1	−0.33	−13.8
Diseases of respiratory system	0.39	0.65	0.62	0.27	14.7	−0.03	−5.8	0.24	9.9
Accidents, suicide, violence[a]	1.27	1.45	1.31	0.18	9.8	−0.14	−25.2	0.04	1.7
Motor vehicle accidents	0.33	0.59	0.43	0.26	14.2	−0.16	−28.6	0.10	4.2
Other accidents	0.64	0.54	0.41	−0.11	−5.8	−0.12	−21.9	−0.23	−9.6
Suicide	0.23	0.27	0.37	0.05	2.5	0.10	17.6	0.14	6.1
Diseases of circulatory system[a]	1.15	2.36	2.75	1.22	66.5	0.39	70.1	1.61	67.5
Ischaemic heart disease	0.95	1.76	1.92	0.82	44.7	0.16	28.1	0.97	40.9
Cerebro-vascular disease	0.04	0.27	0.30	0.24	13.1	0.02	4.0	0.26	11.0
Neoplasms[a,b]	0.31	0.85	1.29	0.54	29.5	0.45	80.2	0.99	41.4
Trachea, bronchus, lung	0.28	0.61	0.81	0.33	17.9	0.20	36.2	0.53	22.2
Female breast	−0.28	−0.35	−0.39	−0.07	−4.0	−0.04	−6.7	−0.11	−4.6
Female genital organs[c]	−0.26	−0.21	−0.26	0.05	2.7	−0.05	−9.2	[d]	[d]
Male genital organs[e]	0.13	0.18	0.28	0.06	3.1	0.10	18.0	[d]	[d]
Chronic liver disease and cirrhosis	0.07	0.17	0.24	0.10	5.5	0.07	11.9	0.17	7.0
Symptoms and ill-defined conditions	0.18	0.17	0.18	−0.01	−0.7	0.01	2.0	.00	.0
All other causes	0.78	0.55	0.46	−0.23	−12.6	−0.09	−16.2	−0.32	−13.5

Sources: Calculated from registered deaths by cause and population estimates in the World Health Organization data bank.

NOTE: Sums of details do not in all cases equal totals, because of rounding.

[a] The sums of the details for the sub-categories do not add to the totals shown because the sub-categories are not exhaustive.

[b] The values shown for total neoplasms include malignant and benign neoplasms. The values shown for specific sites are for malignant neoplasms only.

[c] For 1950s and 1970, cancer of cervix and corpus uteri, and placenta. Data for 1980s also include cancer of ovary.

[d] Not computed because contents of category differ substantially for dates being compared (see notes c and e).

[e] For 1950s and 1970s, cancer of prostate only. Data for 1980s also include cancer of testis.

cancer of the trachea, bronchus and lung—the leading cause of male cancer mortality in most developed countries—there was an increase in contribution of 0.5 year (from 0.3 year in the early 1950s to 0.8 year in the early 1980s), based on the means for 23 developed countries. This accounted for about half of the total increase from neoplasms and for about one fifth of the increase from all causes of death. Changes in the contributions of cancer sites that are exclusively male or female (i.e., female breast, female genital organs, male genital organs), were very slight, and tended to offset each other.

In the early 1960s, demographers studying international mortality trends began to notice unfavourable mortality trends among middle-aged and older males in developed countries. The United Nations (1963, pp. 54-96) noted that the declining mortality trends among males over 55 years had been reversed in some developed countries following the Second World War, and that mortality was actually increasing. Similarly, when Coale and Demeny were preparing their regional model life tables in the early 1960s, they observed that in recent life tables for countries in the "West" family, the pattern of male mortality was changing: there was a tendency towards higher death rates above age 50 relative to rates under 30 (Coale and Demeny, 1966, pp. 14, 20).

In a detailed cross-national analysis to determine the underlying causes of the deterioration of older male mortality in the post-War period, Preston (1970) examined several measures of excess male mortality in relation to 10 "explanatory" variables (including two "smoking" variables) in 17 developed countries. The results strongly implicated smoking in the pattern of male mortality change. Those results, together with trend data on deaths by cause and the results of epidemiological studies, led Preston to conclude that cigarette smoking was largely responsible, through increased mortality from circulatory diseases, cancer and bronchitis, for the unfavourable trends in male mortality. Although the contribution of other risk factors was acknowledged (namely, increases in cholesterol consumption, obesity and decreasing exercise levels), their contributions were likely to be small compared to that of smoking (p. 97).

Retherford (1975) related patterns of cigarette smoking in the United States to changes in the sex differential in life expectancy between the ages of 37 and 87 ($_{50}e_{37}$), and concluded that about 75 per cent of the increase in the sex differential between 1910 and 1962 was due to cigarette smoking, and that cardio-vascular diseases and cancer accounted for most of the observed effects (p. 104).

Waldron (1986a) has made estimates of the contribution of cigarette smoking to the sex differential in mortality (as measured by death rates rather than life expectancy), based on data from seven studies conducted between 1948 and 1980. Six of the studies were in the United States, and the seventh in Sweden. By comparing sex differentials in mortality among non-smokers with sex differentials in mortality in the total population, Waldron estimated that the proportion of the sex differential attributable to smoking in the adult population was between 40 and 60 per cent. The effects of smoking appeared to decrease with age, from about two thirds of the sex differential in mortality at age 40 to about one fourth at age 80. Data from a few of the studies permitted the estimation of the contribution of smoking to the sex differential in mortality for two causes of death through which smoking claims most of its victims—lung cancer and ischaemic heart disease. For lung cancer, about 90 per cent of the sex differential in mortality appeared to be attributable to smoking, while for ischaemic heart disease the proportion decreased with age, from 60 per cent at ages 45-54 to 18 per cent at ages 75-84. The author cautions that the single-factor analysis she employed probably yields higher estimates of the contribution of smoking to the sex mortality differential than would be obtained from a multivariate analysis that took other relevant factors into account.

3. *Accidents and violence*

The means for the five countries (tables 19-23) show little change in the contribution of accidents and violence (including suicide and homicide) to the sex differential in life expectancy in the twentieth century. The mean contribution increased slightly from 1.1 years in the early 1900s to 1.3 years in 1970, then declined to reach 1.1 years again in the early 1980s. However, trends varied among the five countries, with contributions increasing by 0.6 year in Italy between the 1900s and 1980s and by 0.4 year in the United States, and decreasing by 0.2 to 0.4 year in the other countries.

Although the mean contributions from accidents and violence together did not change much in the five countries from the 1900s to the 1980s, there were compensating changes between the two components of this group that are shown separately. The contribution of motor vehicle accidents increased, while the contribution of the combined category of other accidents, suicide and homicide decreased. The mean contribution from motor vehicle accidents increased from 0 in the 1900s to 0.5 year in 1951, to 0.6 year in 1970, then declined to 0.4 year in the 1980s. On the other hand, for "other accidents and violence", the means declined from 1.1 years in the 1900s to 0.8 year in the 1950s, to 0.7 year in the early 1980s. Prior to the 1950s, the data do not permit a more detailed analysis by cause for "other accidents and violence", but this category includes components that may have displayed opposing trends in their contributions to the sex differential in life expectancy.

The means for the 23 countries in table 24 for all accidents and violence show trends similar to those of the five countries between the 1950s and 1980s, but the values are slightly higher. The mean contribution from accidents and violence increased from 1.3 years in the 1950s to 1.5 years in 1970, then declined to 1.3 years in the early 1980s. The trends in contributions from motor vehicle accidents are also similar to those of the five countries. They show an increase from 0.3 year (1950s) to 0.6 year (1970), then a decline to 0.4 year (early 1980s). In contrast to the averages for the five countries, the category "all other accidents" for the 23 countries

excludes suicide and homicide. The contribution from "all other accidents" declines from 0.6 year in the 1950s to 0.4 year in the 1980s. The contribution of suicide, on the other hand, increases slightly.

The relative (to female) improvement in male mortality from accidents and violence that is reflected in the negative contribution of "other accidents" to the changing sex differential in life expectancy between the 1900s and the 1980s is due to a combination of factors that includes declining employment in hazardous occupations and improved standards of industrial safety, both of which would benefit males more than females (Lopez, 1983, pp. 90 and 110). In the United States, to take an example, the "industrial" death rate declined from 15.7 per 100,000 in 1932 to 2.4 in 1978 (Chesnais, 1985, p. 269).

4. *Infectious and parasitic diseases and certain other diseases*

The causes of death that comprise group I in tables 19 to 23 include the infectious and parasitic diseases; pneumonia, influenza and bronchitis; diarrhoeal disease; and certain diseases of infancy. With the exception of maternal mortality, these are the same causes that comprised "group I" in table 15. Maternal mortality was included with the group I causes in table 15, but is shown separately in tables 19 to 23. Mortality from these causes was high in developed countries early in the twentieth century, but very large mortality declines have occurred during the transition to low mortality.

Of the six groups of causes in tables 19 to 23, around 1900, group I made either the largest or the second largest contribution (after accidents, suicide and violence) to the sex differential in life expectancy, with contributions ranging from 2.8 years in England and Wales (1901) down to 0.5 year in Italy (1901), and averaging 1.4 years in the five countries. As mortality from the group I causes declined, their contribution to the sex differential in life expectancy also declined in four of the five countries (the exception was Italy). By the early 1980s the mean contribution from group I causes in the five countries had declined to 0.6 year, with a range from 0.3 year in the United States to 0.9 year in England and Wales. The trends therefore exerted a narrowing effect on the sex differential that averaged −0.8 year for the five countries between around 1900 and the early 1980s (half of this negative contribution was made between 1900 and the 1950s, and half between the 1950s and 1980s). It should be noted that the inclusion of some categories of chronic respiratory diseases with the group I causes, particularly in 1970 and the 1980s (when, in addition to bronchitis, emphysema and asthma were also included), tends to mitigate the narrowing effect on the sex differential exerted by this group of causes. Had it been possible to consistently exclude the chronic respiratory diseases for all time periods, the narrowing effect contributed by the group I causes would have exceeded −0.8 year.

5. *Maternal mortality*

Maternal mortality refers to female mortality associated directly or indirectly with abortion, pregnancy and childbirth. Around the turn of the century, maternal mortality made a negative contribution to the sex differential in life expectancy that averaged −0.4 year in the five countries (tables 19-23). As a result of extremely large declines in maternal mortality, the negative contributions from this cause have been virtually eliminated; consequently, trends in maternal mortality have resulted in a widening of the sex differential by a mean of 0.4 year in the five countries.

The following table illustrates the magnitude of the declines in maternal mortality rates in those countries. The rates shown are the number of deaths from maternal causes per 100,000 female population in the reproductive ages (15-49 years), unadjusted for differences in age structure among the five populations. It should be noted that maternal mortality rates can also be related to the number of births, depending on the purpose of the analysis.

MATERNAL MORTALITY RATES, FIVE DEVELOPED COUNTRIES 1900 AND 1980S

Country	1900s		1980s	
	Year	Rate[a]	Year	Rate[a]
Australia	1911	51.5	1983	0.2
England and Wales (UK)	1901	49.1	1982	0.4
Italy	1901	35.3	1980	0.6
Sweden	1911	26.2	1984	0.1
United States	1900	48.8	1982	0.5

Source: Calculated from Preston, Keyfitz and Schoen (1972) and World Health Organization data bank.

[a] Maternal deaths per 100,000 female population aged 15-49.

IV. SUMMARY

Estimates were presented on the contribution of age groups and causes of death to sex differentials in life expectancy at birth in most developed countries in the early 1980s, and to the widening of sex differentials in life expectancy in selected developed countries since the turn of the century.

Of the mean sex differential in life expectancy of 6.7 or 6.8 years in the early 1980s, diseases of the circulatory system accounted for nearly 40 per cent; neoplasms for 18 per cent; accidents, suicide and violence for 19 per cent; and diseases of the respiratory system for nearly 10 per cent.

Around the turn of the century, sex differentials in life expectancy were generally narrower by several years than they are today. They began to widen at different times in different countries. The widening was not the result of changes in one or two causes of death, but rather the net effect of many changes. A few of those changes exerted a narrowing force on the sex differential in life expectancy, but the majority had the opposite effect. The widening of sex differentials in life expectancy must be viewed in the context of the profound mortality changes in the twentieth century.

Infectious and parasitic diseases. Large declines have occurred in death rates from infectious and parasitic diseases in the twentieth century. Because male mortality from this group of causes was higher than female mortality, their decline in importance has had a narrowing

effect on the sex differential in life expectancy during the observation period of the present study, for most of the countries examined.

Maternal mortality. Declines in female mortality associated with pregnancy and childbirth have contributed to a widening of the sex differential by several tenths of a year.

Neoplasms. Around 1900, mortality from neoplasms was generally higher for females than for males in developed countries. Mortality from neoplasms has increased much more rapidly for males than for females, particularly for cancers of the respiratory system and other cancers associated with cigarette smoking, resulting in a sizeable contribution from neoplasms to the widening of the sex differential in life expectancy.

Diseases of the circulatory system. Several types of changes have occurred with regard to this group of causes, and all have served to widen the sex differential in life expectancy. Mortality from rheumatic fever, which is infectious in origin and for which female mortality was equal to or higher than that of males, has decreased. Mortality from cerebro-vascular disease, which affected the sexes nearly equally, has also decreased in importance. On the other hand, mortality from coronary artery disease (ischaemic heart disease), which is much more common among males, has increased in importance.

Several studies on the underlying causes of the widening sex differentials in life expectancy were briefly summarized. According to those studies, the single factor that has probably contributed the most to the widening of the sex differential has been the adoption of the cigarette habit by men in large numbers. The contribution of this factor has mainly been through elevated male mortality from lung cancer and heart disease.

Notes

[1] For a description of the World Health Organization files, see World Health Organization, 1985, pp. 522-529.

[2] The formula employed for the decomposition by age group are as follows:

For the first age group:

$$ {}_1\Delta_0 = (e_0^f - e_0^m) - \left[(e_1^f - e_0^m) \frac{(l_1^f + l_1^m)}{2} \right] $$

For ages 1-14, 15-24, . . . , 55-74:

$$ {}_n\Delta_x = \left[(e_x^f - e_x^m) \frac{(l_x^f + l_x^m)}{2} \right] - \left[(e_{x+n}^f - e_{x+n}^m) \frac{(l_{x+n}^f + l_{x+n}^m)}{2} \right] $$

For the open-ended age group (75 and over):

$$ {}_\infty\Delta_{75} = (e_{75}^f - e_{75}^m) \frac{(l_{75}^f + l_{75}^m)}{2} $$

Where ${}_n\Delta_x$ is the contribution to the sex differential in life expectancy at birth of mortality differences within age group (x, x+n); n is the length of the age interval; $e_x^f(e_x^m)$ is life expectancy at age x for females (males); $l_x^f(l_x^m)$ measures the life table survivors to age x for females (males) in a life table with a radix of 1 (it also measures the probability of surviving from birth to age x) (see United Nations, 1982b, p. 11 and 1983, p. 27).

[3] The formulae employed for the decomposition by cause of death are as follows:

For each age group (x, x+n), the contribution of cause (i) to the sex differential in life expectancy at birth was obtained from the formula

$$ \frac{r_i^m - r_i^f}{r^m - r^f} {}_n\Delta_x $$

where $r_i^m(r_i^f)$ is the male (female) death rate from cause (i) at age (x, x+n); $r^m(r^f)$ is the male (female) death rate from all causes at age (x, x+n); ${}_n\Delta_x$ is the contribution of age group (x, x+n) to the sex differential in life expectancy at birth.

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ANNEX: Exact time periods for data shown in table 12

Region and country	*Exact time periods for dates before 1950*	*Exact time periods for dates after 1950 if different from dates in column headings*
Northern America		
Canada	1926-1930; 1930-1932	
United States	1900-1902; 1909-1911; 1919-1921; 1929-1931	1980-1983
East Asia		
Japan	1899-1903; 1909-1913; 1926-1930; 1935/36	
Europe		
Eastern Europe		
Bulgaria	1906-1912; 1925-1928; 1933-1936	1955-1959
Czechoslavakia	1899-1902; 1909-1912; 1929-1932	1955-1959
German Democratic Republic		Average for 1980, 1981, 1983, 1984
Hungary	1900-1901; 1920-1921; 1930-1931	
Poland	1931-1932	
Northern Europe		
Denmark	1901-1905; 1911-1915; 1921-1925; 1931-1935	
Finland	1881-1890; 1901-1905; 1921-1930; 1931-1935	
Ireland	1900-1902; 1910-1912; 1925-1927; 1935-1937	Average for 1980, 1981, 1983
Norway	1856-1865; 1901-1905; 1910; 1920; 1930	
Sweden	1851-1860; 1901-1910; 1911; 1920; 1931-1940	
United Kingdom		
England and Wales	1871-1880; 1901-1910; 1910-1912; 1920-1922; 1930-1932	
Northern Ireland	1925-1927; 1936-1938	1980-1982
Scotland	1910-1912; 1920-1922; 1930-1932	
Southern Europe		
Greece		1950-1952; Average for 1980-1982 and 1984
Italy	1881-1882; 1899-1902; 1910-1912; 1921; 1931	1980-1981
Portugal	1919-1922; 1929-1932	Average for 1980-1982 and 1984
Spain	1900; 1910; 1920; 1930	1950; 1965-1969
Yugoslavia		1980-1982
Western Europe		
Austria	1900-1904; 1930-1933	
Belgium	1880-1890; 1928-1932	Average for 1980-1982 and 1984
France	1875-1877; 1898-1903; 1908-1913; 1920-1923; 1928-1933	
Netherlands	1870-1879; 1900-1909; 1910-1920; 1921-1930; 1931-1940	
Switzerland	1876-1880; 1901-1910; 1921-1930; 1930	
Oceania		
Australia-New Zealand		
Australia	1881-1890; 1901-1910; 1911; 1920-1922; 1932-1934	
New Zealand	1880-1892; 1901-1905; 1911-1915; 1925-1927; 1934-1938	
USSR	1896-1897; 1926-1927; 1938-1939	1955-1956; 1971-1972; 1983-1984; 1984-1985; 1985/86

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